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OBSERVER VARIATION IN THE INTERPRETATION OF PARANASAL SINUS SKIAGRAMS

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As Cochrane and Garland (1952) have remarked, there has recently been an increasing interest in methods of measuring, and, if possible, reducing, the effect of observer error in fields where its existence has previously been ignored. In the radiological field, these workers, along with Birkelo, *et al.* (1947), Yerushalmy, *et al.* (1950), Clayson, *et al.* (1955) and others, have investigated observer variation in the diagnosis of pulmonary tuberculosis and found it to be considerable. Fletcher and Oldham (1949) have also noted considerable variation in the radiological diagnosis of pneumoconiosis. More recently, Gandevia and Stradling (1958) have investigated observer variation in the bronchographic diagnosis of chronic bronchitis and of bronchiectasis, and Bull, *et al.* (1960) have investigated observer variation in the reading of cerebral angiograms.

The purpose of the present paper is to present data on observer variation in the evaluation of skiagrams of the paranasal sinuses. We can find no evidence in the literature that this has been done previously. In common with Yerushalmy, *et al.* (1950), Gandevia and Stradling (1958) and others we have used the term "observer variation" instead of "observer error". Use of the first term avoids the necessity of determining the true diagnosis, which may be impossible.

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Procedure

Material and Method

The material on which this study is based was provided by a set of occipito-mental skull views taken during the E.N.T. examination of a random sample of a rural population in the Vale of Glamorgan (South Wales). The survey was designed to define certain E.N.T. characteristics of such a population in the age range 18-74 years inclusive and was stratified for age and sex. For purposes not related to the rhinological aspects of the survey, the sample size of the youngest age group for both sexes was increased as compared with other ages.

A sample of 400 was drawn and we succeeded in X-raying about 90 per cent. of these people.

The films were taken with a 65 Kv. Newton Victor Model F portable X-ray unit. A special cone was made to prevent excessive radiation and scatter reaching the patient. The X-ray apparatus, including the cassette holder, was in a fixed position in a mobile examination unit. All positioning adjustments had to be made by moving the subject. It was realized that, because of these deficiences and the necessity of a long exposure time (about 5 seconds), the quality of the films would not be as high as that encountered in hospital practice. The advantage of this procedure, however, was that a subsequent classification of the films would enable us to assess the influence of technical quality on observer variation. The survey was also designed to explore the potentialities of radiography of the paranasal sinuses as a possible index in epidemiological research.

Two films were taken for each subject, identified by number only, and, when processed, placed in a single envelope. One film was taken at the conventional 45° tilt of the radiological base line, the other film at a slightly greater angle.

Reading the Films

The films were arranged in a haphazard order and independently read by four observers. There was no prior discussion between the observers, nor any consultation between them when the films were being read. Each observer was allowed to select and report on the better of the two films for each subject. All observers used the same General Electric "Fluoroline" illuminator fitted with masks and reported on a proforma (see Table I). Of the four observers, only one (a radiologist) reported on "Technique" in addition to "Appearances". A fifth observer, a radiographer, also reported on "Technique", but not on "Appearances". Each of the four who read for "Appearances", however, was allowed to decide whether the film was "Unreadable" or "Readable".

An attempt was made to evaluate intra-observer variation by, unknown to the observer, taking 20 envelopes of films that had been read

TABLE I.

REPORTING SHEET FOR THE PARANASAL SINUS SKIAGRAMS.

OCCIPITO-MENTAL SKULL VIEWS.

Film No..... Reader..... Date.....

TECHNIQUE.

Acceptable	Fair	Unreadable	Faults
			Movement
			Positioning
			Over Angulation
			Under Angulation
			Rotation
			Exposure
			Over
			Under
			Other
			(Specify)

APPEARANCES

	Right Yes	Right No	Left Yes	Left No
(A) *SINUSES*				
1. Presence of:				
(i) maxillary				
(ii) frontals				
(iii) ethmoids				
2. OPACITY (a) generalized—maxillary				
frontal				
ethmoidal				
(b) localized*				
(i) mucosal thickening				
(ii) fluid level				
(iii) cyst/polyp				
(iv) osteoma				
(v) F.B.				
(B) *MASTOIDS*: Cellularity				

COMMENTS:

* If present, please specify which sinus, i.e. M.(=maxillary), F(=frontal), E(=ethmoidal), against appropriate abnormality.

and placing them at the bottom of the "unread" pile. We were able to do this in the case of one observer only, viz. Observer "A". This sample of twenty was drawn haphazardly from the "read" pile.

The Observers

The differing experience of the observers is indicated by the following notes:

"A" An otolaryngologist with about 10 years experience in the speciality.

"B" An otolaryngologist with at least 20 years experience in the speciality.

"C" A radiologist with about 3 years experience in the speciality.

"D" A doctor with about 2 years experience in otolaryngology.

"E" A radiographer with considerable experience with radiological methods in epidemiology, who reported on technique only.

Results

All four observers agreed that there were " readable" films in a total of 285 cases out of 358 individuals X-rayed. Two women in the total sample of 360 refused X-ray examination on the grounds that they believed it to be hazardous. A varying number of cases were considered to have "unreadable" films, or no report was made, by one or more of the observers. In only 3 cases, however, were the films considered to be unreadable by all four observers. This total of 285 cases—or 570 sides when right and left are considered independently—forms the basis for our analysis. The results are presented in a manner similar to that used by Gandevia and Stradling (1958). Tables II-XIII inclusive indicate the

TABLES II-VII INCLUSIVE.

TABLES INDICATING THE OBSERVER VARIATION IN THE READING OF PARANASAL SKIAGRAMS.

MAXILLARY SINUSES

OBS 'A'	PREPONDERANT DIAGNOSIS: NORMAL	GENERALISED OPACITY	FLUID LEVEL	MUCOSAL THICKENING	CYST OR POLYP	F.B.	TOTAL
INDIVIDUAL DIAGNOSIS: NORMAL	414	0	2	5	1	2	424
GENERALISED OPACITY	8	19	0	5	0	0	32
FLUID LEVEL	1	0	2	1	0	0	4
MUCOSAL THICKENING	61	1	0	40	4	0	106
CYST OR POLYP	0	0	0	0	4	0	4
F.B.	0	0	0	0	0	0	0
TOTAL	484	20	4	51	9	2	570

TABLE II.

MAXILLARY SINUSES

OBS 'B'	PREPONDERANT DIAGNOSIS: NORMAL	GENERALISED OPACITY	FLUID LEVEL	MUCOSAL THICKENING	CYST OR POLYP	F.B.	TOTAL
INDIVIDUAL DIAGNOSIS: NORMAL	473	0	0	8	1	2	481
GENERALISED OPACITY	2	19	0	2	0	0	23
FLUID LEVEL	0	0	3	0	0	0	3
MUCOSAL THICKENING	9	1	1	41	1	0	53
CYST OR POLYP	0	0	0	0	4	0	0
F.B.	0	0	0	0	0	0	0
TOTAL	484	20	4	51	9	2	570

TABLE III.

MAXILLARY SINUSES

OBS. 'C'

INDIVIDUAL DIAGNOSIS	PREPONDERANT DIAGNOSIS: NORMAL	GENERALISED OPACITY	FLUID LEVEL	MUCOSAL THICKENING	CYST OR POLYP	F.B.	TOTAL
NORMAL	471	3	0	22	0	0	496
GENERALISED OPACITY	0	14	0	2	0	0	16
FLUID LEVEL	0	0	3	0	0	0	3
MUCOSAL THICKENING	10	3	1	22	0	0	36
CYST OR POLYP	3	0	0	5	9	0	17
F.B.	0	0	0	0	0	2	2
TOTAL	484	20	4	51	9	2	570

TABLE IV.

MAXILLARY SINUSES

OBS. 'D'

INDIVIDUAL DIAGNOSIS	PREPONDERANT DIAGNOSIS: NORMAL	GENERALISED OPACITY	FLUID LEVEL	MUCOSAL THICKENING	CYST OR POLYP	F.B.	TOTAL
NORMAL	368	0	2	8	1	0	379
GENERALISED OPACITY	34	20	0	20	1	0	75
FLUID LEVEL	2	0	1	0	0	0	3
MUCOSAL THICKENING	75	0	1	23	0	0	99
CYST OR POLYP	1	0	0	0	7	0	8
F.B.	4	0	0	0	0	2	6
TOTAL	484	20	4	51	9	2	570

TABLE V.

FRONTAL SINUSES

OBS. 'A'

INDIVIDUAL DIAGNOSIS	PREPONDERANT DIAGNOSIS: NORMAL	ABSENT	GENERALISED OPACITY	MUCOSAL THICKENING	OSTEOMA	TOTAL
NORMAL	508	8	1	0	0	517
ABSENT	4	42	0	0	0	46
GENERALISED OPACITY	2	0	1	0	0	3
MUCOSAL THICKENING	1	0	0	1	0	2
OSTEOMA	1	0	0	0	1	2
TOTAL	516	50	2	1	1	570

TABLE VI.

FRONTAL SINUSES

OBS. 'B'

INDIVIDUAL DIAGNOSIS	PREPONDERANT DIAGNOSIS: NORMAL	ABSENT	GENERALISED OPACITY	MUCOSAL THICKENING	OSTEOMA	TOTAL
NORMAL	506	8	0	0	1	515
ABSENT	3	42	0	0	0	45
GENERALISED OPACITY	0	0	1	0	0	1
MUCOSAL THICKENING	7	0	1	1	0	9
OSTEOMA	0	0	0	0	0	0
TOTAL	516	50	2	1	1	570

TABLE VII.

inter-observer variations for observers "A", "B", "C" and "D", and Tables XIV-XVI inclusive show the intra-observer variation for observer "A". The diagonal lines indicate where agreement exists, i.e. where there is no variation. With the exception of squares for "Totals", squares not bisected by the diagonal line indicate a disagreement, the nature of which is indicated by the heading of the row and the column concerned.

In the assessment of interindividual variation, the "correct" diagnosis for a given sinus was considered to be that diagnosis which preponderated amongst the opinions of the four observers, i.e. the modal opinion. If two or four diagnoses were equi-prevalent, a "casting vote" was given by the

author after inspection of the films and a consideration of the contending diagnoses. This "casting vote" was required on 48 (=2·8 per cent.) occasions.

The preponderant diagnosis may not have been the correct diagnosis, since the observer with the greatest skill may have recognized an abnormality that the others had missed. The preponderant diagnosis does, however, serve as a base line for comparing the performance of observers. At the same time, we believe that the preponderant diagnoses do, in this instance, come quite closely to being the true diagnosis.

TABLES VIII-XIII.

TABLES INDICATING THE OBSERVER VARIATION IN THE READING OF PARANASAL SKIAGRAMS.

FRONTAL SINUSES

OBS. 'C' — INDIVIDUAL DIAGNOSIS	PREPONDERANT DIAGNOSIS: NORMAL	ABSENT	GENERALISED OPACITY	MUCOSAL THICKENING	OSTEOMA	TOTAL
NORMAL	511	4	0	0	0	515
ABSENT	2	46	0	0	0	48
GENERALISED OPACITY	3	0	2	1	0	6
MUCOSAL THICKENING	0	0	0	0	0	0
OSTEOMA	0	0	0	0	1	1
TOTAL	516	50	2	1	1	570

TABLE VIII.

FRONTAL SINUSES

OBS. 'D' — INDIVIDUAL DIAGNOSIS	PREPONDERANT DIAGNOSIS: NORMAL	ABSENT	GENERALISED OPACITY	MUCOSAL THICKENING	OSTEOMA	TOTAL
NORMAL	498	5	0	1	1	505
ABSENT	1	45	0	0	0	46
GENERALISED OPACITY	16	0	2	0	0	18
MUCOSAL THICKENING	0	0	0	0	0	0
OSTEOMA	1	0	0	0	0	1
TOTAL	516	50	2	1	1	570

TABLE IX.

ETHMOIDAL SINUSES

OBS. 'A' — INDIVIDUAL DIAGNOSIS	PREPONDERANT DIAGNOSIS: NORMAL	ABSENT	GENERALISED OPACITY	TOTAL
NORMAL	566	1	0	567
ABSENT	0	0	0	0
GENERALISED OPACITY	2	0	1	3
TOTAL	568	1	1	570

TABLE X.

ETHMOIDAL SINUSES

OBS. 'B' — INDIVIDUAL DIAGNOSIS	PREPONDERANT DIAGNOSIS: NORMAL	ABSENT	GENERALISED OPACITY	TOTAL
NORMAL	567	0	0	567
ABSENT	1	1	0	2
GENERALISED OPACITY	0	0	1	1
TOTAL	568	1	1	570

TABLE XI.

ETHMOIDAL SINUSES					
OBS. 'C'		PREPONDERANT DIAGNOSIS			
		NORMAL	ABSENT	GENERALISED OPACITY	TOTAL
INDIVIDUAL DIAGNOSIS	NORMAL	564	1	0	565
	ABSENT	0	0	0	0
	GENERALISED OPACITY	4	0	1	5
	TOTAL	568	1	1	570

TABLE XII.

ETHMOIDAL SINUSES					
OBS. 'D'		PREPONDERANT DIAGNOSIS			
		NORMAL	ABSENT	GENERALISED OPACITY	TOTAL
INDIVIDUAL DIAGNOSIS	NORMAL	454	0	0	454
	ABSENT	104	1	0	105
	GENERALISED OPACITY	10	0	1	11
	TOTAL	568	1	1	570

TABLE XIII.

Discussion

Yerushalmy, *et al.* (1950) concluded from their study of chest survey photofluorograms that observer variation is of disturbing magnitude. Tables II-XIII show that this statement is equally applicable to survey X-rays of the paranasal sinuses. Depending upon the observer, the prevalence of maxillary sinus abnormalities may vary by a factor of 2·3. Yerushalmy *et al.* (1950) found that the prevalence of "positive" chest photofluorograms in their series might vary by a factor of 2·2. Similarly, Yerushalmy found that the probability of a single reader missing a "positive" film was of the order of 0·3, which appears to be the same order of probability of a single reader missing or incorrectly interpreting a given abnormality of the maxillary sinus. The probability of "missing" an absent frontal sinus is lower (about 0·1). Yerushalmy's studies showed that the probability of a false positive diagnosis was somewhat less than 0·02, which is lower than the probability of a false positive diagnosis of a maxillary sinus abnormality in this series (p=0·02-0·24 according to the observer).

Bias of Observers

Inspection of the charted results shows that there is a tendency of observer "A" to "over-read" for mucosal thickening of the antra, a tendency of "C" to "under-read" for this abnormality, and a marked tendency of "D" to "over-read" for both mucosal thickening and generalized opacity of the antrum, as well as regarding the presence of a foreign body in this sinus. A comparison of "A's" tendency to "over-read" with

TABLES XIV-XVI INCLUSIVE.

TABLES INDICATING THE INTRA-OBSERVER VARIATION IN THE READING OF PARANASAL SINUS SKIAGRAMS.

MAXILLARY SINUSES

OBS. 'A' FIRST READING	SECOND READING: NORMAL	GENERALISED OPACITY	MUCOSAL THICKENING	TOTAL
NORMAL	21	0	0	21
GENERALISED OPACITY	0	8	0	8
MUCOSAL THICKENING	3	0	6	9
TOTAL	24	8	6	38

TABLE XIV.

FRONTAL SINUSES

OBS. 'A' FIRST READING	SECOND READING: NORMAL	ABSENT	GENERALISED OPACITY	TOTAL
NORMAL	29	0	0	29
ABSENT	2	6	0	8
GENERALISED OPACITY	0	0	1	1
TOTAL	31	6	1	38

TABLE XV.

ETHMOIDS

OBS. 'A' FIRST READING	SECOND READING: NORMAL	GENERALISED OPACITY	TOTAL
NORMAL	37	0	37
GENERALISED OPACITY	0	1	1
TOTAL	37	1	38

TABLE XVI.

"C's" tendency to "under-read" for mucosal thickening of the maxillary sinuses showed that these biases can be statistically significant. These biases are probably dependent upon an observer's criteria for accepting a given abnormality. Inter-observer differences in criteria will probably be mainly responsible for inter-observer variations, whilst the dependence of such criteria upon mental images will produce an intra-observer variation. One must remember that the errors in Tables II-XIII will reflect not only the "net" inter-observer variations but also the intra-observer variations.

Skill and Experience

As a measure of an observer's skill in reading this set of X-rays, we can add together the data for the three groups of sinuses, i.e. maxillary, ethmoids and frontal, and calculate the percentage agreement of the observer with the preponderant diagnosis. Table XVII shows these percentages alongside the number of years experience that the given observer had had in reading sinus X-rays. The data indicate a correlation coefficient of the order of 0·7. Skill in reading the X-rays is therefore dependent to a large extent upon experience.

TABLE XVII.

TABLE INDICATING SKILL IN READING PARANASAL SINUS SKIAGRAMS AS A FUNCTION OF EXPERIENCE.

Observer	% Agreement with Preponderant Diagnosis	Years of Experience
	%	
"A"	94	10
"B"	97	20
"C"	96	3
"D"	83	2

Quality of the film: Effect of radiographic technique

Observer "C", the radiologist, commented that the general standard of the films was below that of the ordinary X-ray Department, and consequently doubted the general applicability of the findings. It was therefore imperative that we enquired into the influence of the quality of the films. For this purpose, we classified the films that had been reported on by the four observers into two groups, i.e. "Better" quality films and "Poorer" quality films. Remarks on the technique that were made by observers "C" and "E" were used for this classification. Films that were considered "Acceptable" in every respect by both these observers were classified as "better" quality films. Other films were placed in the "poorer" category. For each of the four observers and for each of the two categories, we calculated the percentage of cases in which the observer's diagnosis did not concur with the preponderant diagnosis. For convenience, we considered the reports on the maxillary sinuses only. These results are shown in Table XVIII. Observers "A", "C" and "D" showed a smaller variation with the better quality films. The larger variation that "B" showed with the better quality films is presumably a chance fluctuation since the difference is not statistically significant. A statistically significant difference (χ^2 with Yates' correction=6·2) occurs only in the case of "A". We must conclude that the quality of the films may or may not influence a reader's observer error, depending on the particular

reader. It is to be noted that the observer who was not influenced by the technical quality of the films, i.e. "B", was the one with the greatest experience. It is possible that, the greater the experience, the less is the observer's criteria disturbed by technical faults in films.

TABLE XVIII

TABLE SHOWING THE INFLUENCE OF TECHNICAL QUALITY ON OBSERVER VARIATION IN THE READING OF PARANASAL SINUS SKIAGRAMS.

Observer	% Non-Concurrence with Preponderant Diagnosis (Maxillary Sinuses only)	
	"Poorer" Quality Films	"Better" Quality Films
	%	%
"A"	18·8	9·8
"B"	4·8	6·7
"C"	9·5	6·1
"D"	26·9	22·6

By considering the preponderant diagnosis, we find that the proportion of "sides" with abnormalities of the maxillary sinus is 14 per cent. amongst the better quality films. The proportion in the total group is 15 per cent. This suggests that (a) the make-up of the "better" quality sample is similar to that of the "poorer" quality sample, and (b) technical quality of the films does not influence the reported prevalence of sinus abnormalities. In contrast, Fletcher and Oldham (1949) have demonstrated that, in the case of chest films showing pneumoconiosis, the classification is influenced by the technical quality of the films.

It is to be noted that, even with the present radiographic equipment, less than 1 per cent. of the films were considered unreadable by all four observers. Improved equipment would decrease this rejection percentage to a negligible value.

Intra-Observer Variation

Tables XIV-XVI show that there was "non-concurrence" of the diagnosis in 4·4 per cent. of cases. This is a measure of the intra-observer variation for observer "A". For the same observer, Tables II, VI and X show that there was no agreement with the preponderant diagnosis in 6·5 per cent. of cases. This is a measure of this observer's combined intra- and inter-observer variation. It would therefore appear that the "net" inter-observer variation is smaller than the intra-observer variation. In fact, we might question the existence of any distinct inter-observer variation when we learn that the difference between the two percentages is not statistically significant. We have, however, shown that there is such a thing as observer bias which may be appreciable in its extent. This bias

must introduce a further factor, i.e. inter-observer error into observer variation, in addition to the factor or factors attributable to intra-observer variation.

Analysis of the intra-observer variation in this case is, however, more complicated than it at first seems. First, the proportion of abnormal films in the "intra-observer" sample is significantly greater than in the total, i.e. "inter-observer" sample. It was therefore not a random sample. This might produce an artificial inflation of the intra-observer variation. Secondly, the films that were read by observer "A" a second time were reported on at the end of the total sample. It is therefore possible that, if there is a practice effect in reading films, our results on intra-observer variation are distorted by this factor. Fletcher and Oldham (1949) have demonstrated such an effect, whereby the inter-observer variation appears to decrease. Moreover, on four of the five occasions where "A" gave a different opinion on the second reading, the second opinion concurred with the preponderant diagnosis.

It would seem then that the "net" intra-observer variation, i.e. the time-independent component, might be quite small, perhaps of the order of 2 per cent. (in terms of non-concurrence with the preponderant diagnosis) or less.

"Overlooked" films

It was noted that one or two films had not been reported on at all by some of the observers. It was thought that this might have occurred because the observer had attempted to avoid making a decision on a difficult reading. The number of films that had been "overlooked" in this way might therefore possibly be inversely related to the motivation of the observer. Table XIX shows the number of films that have been "overlooked" together with the observer's "score" which is an index of the

TABLE XIX.

TABLE SHOWING "OVERLOOKED" FILMS AS A FUNCTION OF SKILL.

Observer	% Agreement with Preponderant Diagnosis	Number of Films "Overlooked"
	%	
"A"	94	6
"B"	97	1
"C"	96	5
"D"	83	6

skill of the observer. These results indicated a correlation coefficient of the order of −0·6. It is therefore probable that, the greater the motivation, the greater is the accuracy of the reading. The results, however,

may be interpreted differently. With more years of experience, there are fewer "difficult" films, therefore the number of decisions that are avoided are fewer. Moreover, it might be expected that one or two films might be overlooked by chance.

Conclusions

The inescapable conclusion from this study is that there is a very large observer variation in the reading of X-ray films of the paranasal sinuses. Several factors are responsible for this variation. The predominant factor appears to be observer bias that results in under- or over-reading for a given abnormality and is primarily responsible for inter-observer variations. This bias is probably dependent upon the different criteria that various observers have for judging a given abnormality. To some extent it may be reduced by experience. It indicates, however, the need for systematic training in the reading of paranasal sinus skiagrams and for the investigation of the possibility of mechanical objective aids to consistency in film readings. These conclusions were also reached by Clayson, *et al.* (1955) in respect of chest skiagrams. A joint discussion on a few films by the observers before reading the whole group would most probably have reduced the inter-individual variation appreciably.

With respect to the reading of survey skiagrams, we can conclude that at least three observers should be used and these should have as much experience as possible in this field. We would suggest a minimum of three years' experience in reading sinus skiagrams. Observer "D" clearly had insufficient experience and it might be contended that, on these grounds it was unfair to have employed him. The data that he provided, however, indicated the extent to which experience influences observer variation.

Summary

Using occipito-mental skull views taken on a random sample of a rural population, observer variation in the reading of paranasal sinus skiagrams has been investigated. There is considerable inter-individual variation. This appears to be dependent upon the different criteria that different observers have for judging a given abnormality, and is only partially reducible by experience.

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EPISTAXIS MORTALITY

THE PLACE OF BLOOD TRANSFUSION IN ITS PREVENTION

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NOSE bleeding ceases spontaneously in most cases and is usually a trivial complaint (Hallberg, 1952). It is however a potentially fatal condition and death probably occurs more often than is generally supposed. Although there has from time to time been considerable discussion about the management of epistaxis, this has mainly dealt with the surgical measures necessary to stop the bleeding. Less attention has been paid to the management of the systemic complications of severe epistaxis, i.e. hypotension and anæmia, and to the place of transfusion in their correction. In this paper seven fatal cases are described, with note of the post-mortem findings in five of them. Cardiovascular factors were found to be of considerable importance in all of them.

Case Reports

CASE I.

Mrs. E.H., aged 75 years. This patient was first seen by Mr. J. W. Cope at the Royal National Throat, Nose and Ear Hospital in November 1955. She gave a history of recurrent epistaxis of recent onset and was admitted to hospital for one day. The exact site of bleeding could not be seen but after the nose was packed it ceased. Subsequent epistaxis required frequent readmission. Her diastolic blood pressure was always less than 100 mm. Hg, and there were no signs of systemic disease. In February 1956, her hæmoglobin was found to be 48 per cent. (Haldane) and she was given packed red cells from one pint of blood. Her hæmoglobin rose to 64 per cent. (Haldane) and epistaxis did not recur. A fortnight later she suddenly became stuporous and showed signs of acute heart failure. She died on the following day.

Post-mortem findings: There was extensive atheroma of the coronary arteries and the anterior descending branch of the left coronary artery was blocked by thrombus which had caused a large myocardial infarct. Microscopy showed this to be recent in origin. No local nasal lesion found.

CASE II.

Miss M.G. aged 63 years was a patient of Dr. J. H. Sheldon at the Royal Hospital, Wolverhampton, admitted in March 1956, having been

bleeding severely from the left nostril for an hour. There was no relevant past history. B.P. 130/90. A transfusion on one pint of blood was given and the nose bleed stopped. Eleven days later the nose bleeding recurred and the blood pressure fell to 90/60. Hæmoglobin at this time was 75 per cent. (Haldane). The patient appeared to recover from this episode, but two days later she suddenly collapsed and died three minutes later.

Post-mortem findings: In the heart there was marked left ventricular hypertrophy. The aortic valve was bicuspid and completely calcified with fusion of the cusps at either end. The resultant stenosis must have been of long standing. The other valves were normal and the coronary arteries healthy. No local lesion found in the nose.

CASE III.

Mr. H.G. aged 22 years was admitted to New Cross Hospital, Wolverhampton under the care of Dr. E. J. Blair in July 1956 with a severe epistaxis. He also complained of headache and dizziness for the two previous days. His blood pressure was 210/110. He had albuminuria and a blood urea of 460 mg. per cent. After admission he became comatose and died on the following day.

Post-mortem findings: The brain showed petechial pontine hæmorrhages. The lungs were the site of an acute purulent bronchitis. Both kidneys showed an extreme degree of hydronephrosis. The surfaces were grossly scarred and little renal parenchyma remained. The ureters were both greatly dilated and their openings into the bladder were large and incompetent. No organic obstruction present. It was considered that these lesions were congenital. No local lesion found in the nose.

CASE IV.

Mr. E.P. aged 65 years was admitted to the Royal Hospital, Wolverhampton in October 1956, under the care of Mr. W. L. Thomas following a severe nose bleed. There was no relevant previous history. Blood pressure 180/120. The nose was packed and morphine given but the bleeding could not be stopped despite repacking. A transfusion of ¾ pint of blood was given three days later but on the following day the patient became comatose and died.

Post-mortem findings: There was some coronary atheroma but no other significant lesions. Death was considered to be due to hæmorrhage. No lesion was found in the nose.

CASE V.

Miss A.P., aged 87 was first seen by Dr. C. Evans at St. Charles' Hospital, London in July 1958 when her nose had been bleeding for two days. Her blood pressure was 150/80 and hæmoglobin 58 per cent. (Haldane). She was given morphine and both sides of her nose were packed.

The bleeding stopped and she was sent home three weeks later taking iron tablets and with a hæmoglobin of 63 per cent. (Haldane). Two months later she was again admitted with epistaxis. Her blood pressure was 115/60 and hæmoglobin 53 per cent. (Haldane). Her nose was packed and one pint of blood given. Bleeding stopped and she was discharged after a week in bed. Two weeks later she returned with a further hæmorrhage which did not cease after three days in hospital. Her hæmoglobin was at this time 46 per cent. (Haldane). A bleeding point on the left side was found and cauterized and one pint of blood was transfused and her hæmoglobin rose to 67 per cent. (Haldane). On the next day, however, another hæmorrhage occurred and the blood pressure fell to 90/50. The patient showed marked mental deterioration and died three days later.

Post-mortem findings: There was an acute bronchitis but no other significant lesion. It was considered that death was due to hæmorrhage combined with the effect of a pre-existent post-hæmorrhagic anæmia.

CASE VI.

Mr. S.R. aged 60 years was admitted to St. Charles' Hospital, London under the care of Dr. Hinds-Howell in December 1958. His nose had been bleeding for most of the day and had not been staunched by packing. Blood pressure 146/90. An oozing area on the septum was cauterized and a paraffin pack was inserted. Two days later the nose was still bleeding and further cauterization was carried out. The bleeding stopped and the hæmoglobin at this time was 67 per cent. (Haldane). Four days later the patient complained of a severe pain in the chest radiating down both arms. He was very breathless and his blood pressure fell to 110/60. A diagnosis of coronary thrombosis was made. The patient died two days later. A post-mortem examination was not performed.

CASE VII.

Mrs. E.A. aged 88 years was admitted to St. Charles' Hospital under the care of Dr. C. Evans having had intermittent epistaxis for some weeks. Plugging of the nose had not stopped the bleeding. On admission the patient was pale and confused. There was a little ankle œdema. Blood pressure 160/90. Hæmoglobin 36 per cent (Haldane). A pack was inserted into the nose and bleeding stopped. Two days after admission a transfusion of one pint of blood and one pint of packed red cells was given. The patient remained rather confused and later became semi-conscious and incontinent of urine. She died five days after admission. Death was considered to be due to anæmia following hæmorrhage. A post-mortem examination was not performed.

Epistaxis Mortality

Epistaxis Admissions to Hospital

An attempt has been made to assess the potentially dangerous factors in epistaxis by reviewing one hundred consecutive cases admitted specifically for this cause to the Royal National Throat, Nose and Ear Hospital. Only cases considered severe enough to require admission have been included. Patients in whom the bleeding followed some surgical manœuvre have not been included, nor have those admitted for some reason other than the severity of the bleeding, e.g. children requiring general anæsthesia to allow cautery of the nose.

The age distribution of the group is shown in Table I. The majority of cases occur in middle life or later. There were 53 males and 47 females. There was less than one week's history of nose bleeding in most cases, but eight cases had been bleeding recurrently for a year or more (Table II). One patient in the group died in hospital (Case I).

TABLE I.

AGE DISTRIBUTION OF EPISTAXIS ADMISSIONS TO HOSPITAL

Age (years)	under 21	21-30	31-40	41-50	51-60	61-70	over 70	TOTAL
No. of cases	7	4	7	8	24	24	26	100

TABLE II.

DURATION OF SYMPTOMS IN EPISTAXIS ADMISSIONS.

Duration	1 day or less	1 day-1 week	1 week-1 month	1 month-1 year	1 year-or more	No record	TOTAL
No. of cases	26	39	16	10	8	1	100

A hæmoglobin estimation was performed on admission in 68 cases and a variable degree of anæmia was found in most of them (Fig. 1). In 17 of the 68 cases in which the hæmoglobin was measured it was less than 60 per cent. (Haldane). The presence of anæmia was not related to the duration of symptoms, nor to the age of the patient. The blood pressure was measured on admission in 84 cases and of these 30 had a diastolic pressure greater than 90 mm. Hg. In 8 cases the diastolic blood pressure was greater than 110 mm. Hg.

Five patients gave a history of cardiac disability other than coronary artery disease, and two patients had suffered from myocardial ischæmia. Two patients had acute attacks of coronary thrombosis in hospital following their epistaxis. In one case this was the cause of death. In both these cases bleeding necessitated blood transfusion.

Blood transfusion was given as part of the treatment in 9 cases. All these patients had a hæmoglobin of less than 60 per cent.; 5 were hypertensive. They were all over 50 years old. Two or three pints of blood were given in all cases except the one which later proved fatal.

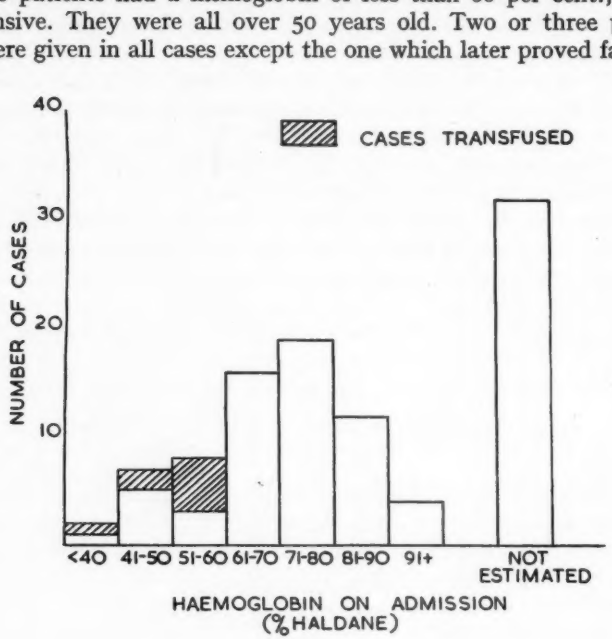

FIG. 1.

Discussion

There is little doubt that the mortality from epistaxis has been seriously underestimated. In the nine years from 1950 to 1958 the Registrar General's records show only 8 deaths from this condition in England and Wales. During a much shorter period, however, we have known of seven fatal cases occurring in only 4 hospitals. A similar impression of the frequency of fatal epistaxis was gained by Collier (1948) who states she had known of 5 such cases.

The reason for this discrepancy is probably that in many cases the immediate cause of death is a complication of the epistaxis, e.g. coronary thrombosis; or there may be a pre-existing disease, most commonly hypertension. These are certified as the cause of death and the epistaxis is not recorded.

An examination of the literature suggests that the majority of these cases are not published. It has been possible to trace only 5 case reports of fatal epistaxis. Of these, the bleeding was secondary to leukæmia (Bourdon, 1952), rupture of a carotid aneurysm (Rasquin, 1949) and

Osler's disease (Siguier, 1954). One developed myocardial infarction due to post-hæmorrhagic hypotension (Bean, 1937) and the fifth patient died of multiple cerebral hæmorrhages following carotid ligation for otherwise uncontrollable bleeding (Hallberg, 1952).

In a condition which is as common as epistaxis, and is so often trivial and self limiting it is difficult for the clinician to pick out those few cases which may develop serious complications, and treat them appropriately. Three factors seems to us important in determining a serious outcome. The first is the degree and duration of post-hæmorrhagic hypotension; the second is the presence of pre-existing heart disease, especially coronary insufficiency; and the third is the presence of anæmia.

Sudden reduction of the blood pressure is well known to be dangerous. Where the blood pressure is high to begin with, even reduction to normal levels can be damaging. Master, *et al.* (1950) analysed 103 cases of acute hæmorrhage and found 59 to show clinical or E.C.G. evidence of coronary insufficiency. Among 13 of their cases which were examined post-mortem 4 showed myocardial necrosis without coronary occlusion. Actual thrombosis is also a recognized complication of profound hypotension. Blumgart *et al.* (1941) found that coronary occlusion was due to hypotension in 11 out of 450 cases, and they emphasize the importance of preventing under any circumstances a decrease of blood pressure in cases of coronary sclerosis. Two out of the 100 Royal National Throat, Nose and Ear Hospital patients developed a coronary thrombosis while in hospital, and one of these died (Case I).

The presence of heart disease in a patient who has a severe epistaxis might be expected to increase the risk, and in fact all the fatal cases but one were suffering from severe cardiovascular disease.

Anæmia of some degree is common in patients with epistaxis (Fig. 1) and may be profound. In anæmia the oxygen carrying capacity of the blood is reduced, with a consequent reduction in the repiratory reserves of the tissue. This is especially important in the myocardium, where signs of disturbed function due to anoxia are often seen. Pickering and Wayne (1934) found anginal symptoms in 8 out of 25 anæmic patients, and Wood (1956) observed E.C.G. changes of an ischæmic character in a similar proportion of cases. Friedberg (1956) is of the opinion that even myocardial infarction can occur due to anæmia alone. It is clear that, if hæmorrhage with consequent hypotension occurs in the presence of anæmia, the oxygen supply to the myocardium becomes critical and infarction is likely to occur.

When more than one of these three factors is present in the same patient the risks will be greater. When all three are present there should be considerable cause for alarm.

The systemic effects of hæmorrhage are in no way related to the site of the bleeding, and the results of a severe epistaxis may be as dramatic

as those following a hæmatemesis. The dangers of hypotension and anæmia in the latter condition are well recognized and blood transfusion is the "sheet anchor of treatment" (Hunt, 1957). This should be equally true in the management of severe epistaxis.

In recent years several authors have emphasized the importance of prompt replacement of blood lost by severe nose bleeding, particularly in patients already suffering from hypertension or coronary disease (Collier, 1948), (Keogh, 1952). In spite of this, current teaching in Otolaryngology does not appear to accept this view. The myth that epistaxis is in some way beneficial to the hypertensive patient ("Nature's safety valve"), still survives in the most recent British textbooks. Negus (1955) says: ". . . In conditions of high arterial tension bleeding from the nose is looked upon as salutary, and not be checked too hastily", and Milling (1953) teaches that: ". . . Epistaxis, particularly in old people with degenerate vessels also requires a guarded prognosis, because if the flow of blood be too promptly arrested, one of the cerebral vessels may be the next to give way." The concern is with the danger of stopping the bleeding too quickly. Neither author mentions the possibility of complications due to hypotension, and only a brief phrase refers to the remote necessity of blood transfusion. Most of the other standard British textbooks present similar views.

In any patient suffering from severe epistaxis it is important to know if heart disease is present. A careful history, paying special attention to such symptoms as angina or dyspnœa on exertion, and past history of coronary disease as well as a full examination of the cardiovascular system should be routine. If there are any positive findings the clinician can then be on his guard.

The other two danger factors, anæmia and hypotension, are both readily treated by blood transfusion. Where fall in blood pressure is the main indication whole blood should be used; if anæmia is being treated packed cells are more appropriate, particularly if heart disease is also present. If these factors are treated vigorously whenever they occur it should be possible to save patients who would otherwise have died from the effects of their epistaxis. It may be noted that, in most of the seven fatal cases, both anæmia and hypotension were left untreated, and even when blood transfusions were given never more than one pint was used. Of the nine cases admitted to the Royal National Throat, Nose and Ear Hospital considered to need transfusion, only one received less than two pints, and this was the only patient in the group who died.

Measures directed toward arresting the hæmorrhage are of prime importance. They have been fully described (Negus, 1955), Hallberg (1952), Collier (1948), Macbeth (1948) and are not the subject of this paper. It is our belief, however, that current teaching in Otolaryngology lays insufficient stress on the importance of blood transfusion in certain

cases of epistaxis. This is reflected by the finding that, in three of our fatal cases, and 32 per cent. of the Royal National Throat, Nose and Ear Hospital series no hæmoglobin estimations were made on admission. Eight out of 17 patients whose hæmoglobin was less than 60 per cent. on admission were given no transfusion; while one patient admitted with a hæmoglobin of 51 per cent. was treated without transfusion for 10 days, until he developed a coronary thrombosis. A transfusion was then given and he recovered.

We conclude that in the treatment of epistaxis the presence of cardiac disease, anæmia or hypotension should be treated as serious prognostic signs. Prompt treatment of the last two by blood tansfusion may do much to reduce the mortality from this condition.

Summary

A review of seven fatal cases of epistaxis, and of one hundred consecutive hospital admissions for this condition indicates that the cardiovascular effects of the hæmorrhage are of crucial importance. Fatal epistaxis is probably not rare, and active treatment of hypertension and anæmia by blood transfusion may be life-saving.

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CEREBROSPINAL OTORRHŒA IN CHRONIC EAR DISEASE

By P. W. R. M. ALBERTI and J. D. K. DAWES (Newcastle-upon-Tyne)

ONE of the many complications of chronic ear disease and its surgical management is cerebrospinal (C.S.F.) otorrhœa. This complication is uncommon but occurs more frequently than is suspected and therefore may remain unrecognized until some more serious complication ensues. A C.S.F. leak from the external auditory meatus indicates that a communication exists between either the ventricular system or the subarachnoid space and the middle-ear cleft.

The purpose of this communication is to review the causes of C.S.F. otorrhœa and to discuss the ætiology, diagnosis and treatment of the condition when associated with chronic ear disease.

Causes of C.S.F. Otorrhœa

The commonest cause of C.S.F. otorrhœa is fracture of the temporal bone. Other causes are congenital abnormalities, surgery and chronic ear disease with its complications.

The pathogenesis and treatment of C.S.F. otorrhœa have recently been reviewed by Ferrer (1960) who also describes a congenital abnormality producing a leak through the internal auditory meatus. Although congenital abnormalities rarely cause a C.S.F. leak one of us (J.D.K.D.) has seen a child with a C.S.F. leak into the tympanum and the eustachian tube due to a congenital absence of the inner ear. She presented with recurrent meningitis and surgical repair of the dehiscence prevented further recurrence. Bony and soft tissue erosions associated with cholesteatoma or the surgery of chronic otitis media may damage the meninges and produce a C.S.F. leak (vide infra).

Anatomy

The temporal bone is widely related to the subarachnoid spaces of the middle and posterior cranial fossae. The inner ear and Trautmann's triangle are closely related to the cisterna pontis lateralis and the cerebellum, whilst three foramina or fissures run between the posterior cranial fossa and the interior of the temporal bone, viz., the internal auditory meatus, the aqueductus cochleae and the aqueductus vestibuli. The subarachnoid space extends into the aqueductus cochleae and is in direct

continuity with the perilymphatic space. The VIIth and VIIIth cranial nerves pass through the cisterna pontis lateralis and lie in subarachnoid spaces, which extend to the fundus of the internal auditory canal where the arachnoid mater blends with the periosteum 8 to 10 mm. inside the internal auditory meatus.

The lateral part of the superior surface of the petrous temporal bone, which is formed by the tegmen, separates the attic, aditus and antrum from the temporal lobe and more medially the tegmen divides the inner ear from the temporal lobe. In 5·4 per cent. of subjects a congenital dehiscence is found in the tegmen (Toynbee cited by Ballance, 1919). We have met with C.S.F. leaks from both the middle and posterior cranial fossae but none associated with a congenital dehiscence of the tegmen.

Aetiology

C.S.F. otorrhœa occurring as a result of chronic destructive middle-ear disease is usually caused by a combination of erosive disease and excisive surgery, although occasionally it may arise as a result of disease extension only. A leak occurring at or immediately following operation is usually precipitated by surgery; a leak developing a considerable time after operation is precipitated by extension of the disease, albeit often in a mastoid cavity which has previously been subjected to extensive surgery.

Cholesteatoma, eroding large areas of bone, can extend far beyond the normal anatomical confines of the middle-ear clett. It is in this sort of case that attempts at complete excision of pathology may produce a subarachnoid fistula. In Case I (see appendix) cholesteatoma had extended to the petrous apex sequestrating the labyrinth and widely eroding the tegmen. An attempt at complete eradication of the disease produced a profuse C.S.F. leak.

When a labyrinthine fistula arising from chronic destructive middle-ear disease produces distressing symptoms, exploration of the middle-ear cleft and occasionally labyrinthectomy is indicated.

In Ménière's disease destruction of one part of the labyrinth seems to suffice but this is not true in chronic middle-ear disease where care must be taken to see that destruction is complete. Clinical observation and animal experiments (Kristensen 1959) have shown that in the labyrinth, as elsewhere in the body, spreading infection may be walled off by inflammatory reaction tissue and subtotal labyrinthectomy may destroy only a limited segment of the organ leaving the remainder as an isolated functioning mechanism. When middle-ear disease is not brought fully under control recurrence is common, so that spread to hitherto undamaged parts and consequent return of symptoms are possible. In Case II total extirpation of the membranous labyrinth at reoperation

produced a brisk C.S.F. leak emerging through either the aqueductus cochleae or more probably the internal auditory meatus.

An occasional cause of C.S.F. leak is surgical injury to the dura mater internal to Trautmann's triangle. Usually this causes no trouble but separation of the dural edges by packing may create a subarachnoid fistula which becomes manifest only after some hours (Case III).

A rare cause of leakage at the time of operation is rupture of the lateral ventricle. Otogenic temporal lobe abscess may produce severe internal hydrocephalus so that intraventricular pressure may greatly exceed the pressure in the abscess; if the abscess is then opened through the cortex the ventricle may rupture through the cavity. McGuckin (1936) described three such cases and discussed the possible mechanism known at that date.

The strut which supports and separates the lateral portions of the temporal and cerebellar lobes lies in the sinodural angle for it forms both the lateral attachment of the tentorium cerebelli, and marks the site of a deep recess between the middle and posterior cranial fossae. Great care should be taken at the time of operation to see that if at all possible a pillar of bone is left in this region, for when either surgery or disease completely destroys the prop a large dural herniation may occur. This problem usually only exists when there has been extensive disease, in the region of a lateral sinus, which has destroyed or has necessitated removal of large areas of the sinus plate. The hernia commonly consists of a part of the temporal lobe together with its dural covering, as the tentorium remains in position in most cases. Sometimes, however, the hernial sac contains both temporal and cerebellar lobes (Case IV). The whole hernial sac then prolapses forwards into the mastoid cavity (Fig. 1).

When further extension of the disease in this region destroys that part of the dura mater forming the hernial sac the weight of the overlying brain may be sufficient to force apart the dural edges and effectively plug the hole which has been created. Only a transient C.S.F. leak will arise, if indeed it occurs at all. Occasionally, however, the hole is not completely plugged and C.S.F. otorrhœa develops. This may be intermittent, occurring only when further erosion temporarily destroys the seal of inflammatory tissue developed at the junction of brain and dura and drying up as new barriers are formed (Case IV).

An erosion in the region of Trautmann's triangle presents a different problem since the medial relation of the bone is the lateral cistern. The cerebellum is widely separated from the dura so that any defect will communicate directly with the subarachnoid space and it is unlikely to be closed by brain tissue, the secretory pressure of the C.S.F. being sufficient to keep the structures apart. In this type of case profuse C.S.F. otorrhœa may develop dramatically and it may be persistent (Case V).

Cholesteatoma sometimes erodes the tegmen alone to such an extent

that a large area of unsupported temporal lobe and dura herniates into the temporal bone. Further extension of the disease may create a dural gap which, as in the case of lesions at the sinodural angle, is effectively

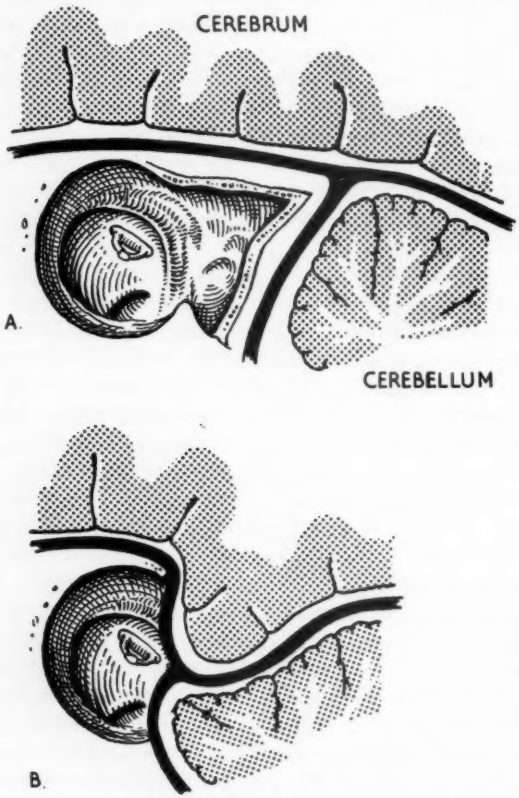

FIG. 1.
Showing (a) how a strut of bone in the region of the sinodural angle supports and separates the temporal lobe from the cerebellum and (b) the resultant hernia if this strut is removed.

plugged by the directly adjacent brain. Occasionally the edges of the defect are not completely sealed, either because the inflammatory barrier has been broken or because the secretory pressure of the C.S.F. is sufficient to keep brain and dura apart (Case VI).

Diagnosis

Diagnosis of C.S.F. otorrhœa presents no difficulty as long as the possibility is borne in mind. The presence of clear fluid in a chronically diseased ear recurring rapidly after toilet should raise the suspicion of

a C.S.F. leak, and it may be confirmed by testing the fluid for sugar, or by seeing if it clots: serum or inflammatory exudate does clot, C.S.F. does not (the handkerchief test). Occasionally a leak is so profuse that the patient, his clothes and the bedding are soaked in C.S.F. No other type of ear discharge is quite so copious. When a fistula is created into the subarachnoid space at operation the presence of clear fluid in the wound tells its own tale.

Antibiotic treatment of an acute exacerbation of chronic suppurative otitis media before a really accurate diagnosis has been made may delay the diagnosis of C.S.F. otorrhœa. These drugs either prevent the onset of meningitis, or quickly clear it; the patient temporarily improves but the C.S.F. leak remains. Antibiotics only help to prevent or cure the infective complications of C.S.F. otorrhœa; they do not alter the presentation of the lesion and they cannot repair the dural defect.

Treatment

The prime need in treatment is the accurate location of the site of the leak. Although in congenital lesions injection of dye may be required, this has not been necessary in our cases of chronic destructive otitis media, where the dural defect has always been evident as soon as the middle-ear cleft has been cleaned. If a small leak has been created at the time of operation in a previously undamaged part, firm packing of the area has seemed to provide adequate control. Temporalis muscle is easily available and works well but gelfoam and B.I.P.P. gauze are also useful (Cases I and II). In fact leakage may be arrested spontaneously.

C.S.F. otorrhœa coming from a dural defect in which dural edges are being held apart by external packing, inflammatory adhesions or the weight of overlying brain does not cease spontaneously and surgical closure is indicated. Any attempt at repair should be preceded or accompanied by excision of the underlying diseased tissues for if this is neglected the C.S.F. leak may recur. Anatomical considerations govern the method to be used in the repair. Craniotomy with approach from above gives the best exposure of middle fossa defects, for a good plane of cleavage is obtainable between the two layers of dura mater so that the herniating brain can be easily seen and dealt with (Schurr, 1960).

If the defect is in Trautmann's triangle and therefore medial to the lateral sinus a transmastoid approach gives the best exposure, for in this region the two layers of dura envelop the lateral sinus and it is difficult to obtain a plane of cleavage by the suboccipital approach.

Fascia or muscle are suitable materials for repair; temporalis muscle is easily obtainable and perfectly adequate if the gap is small; fascia lata is suitable if the defect is large. It is both difficult and unnecessary to suture this material to the dura in a transmastoid repair but care should

be taken to tuck the fascia deep to the edges of the bony defect and then to pack it well into place. When craniotomy is performed the herniating temporal lobe must first be reduced, by diathermy amputation if necessary. The hernial sac is best obliterated with a plug of beaten muscle, temporalis being suitable (Hankinson—personal communication) and the defect should be closed with fascia which is laid between the two dural layers and if possible sutured to the dural edges (Fig. 2).

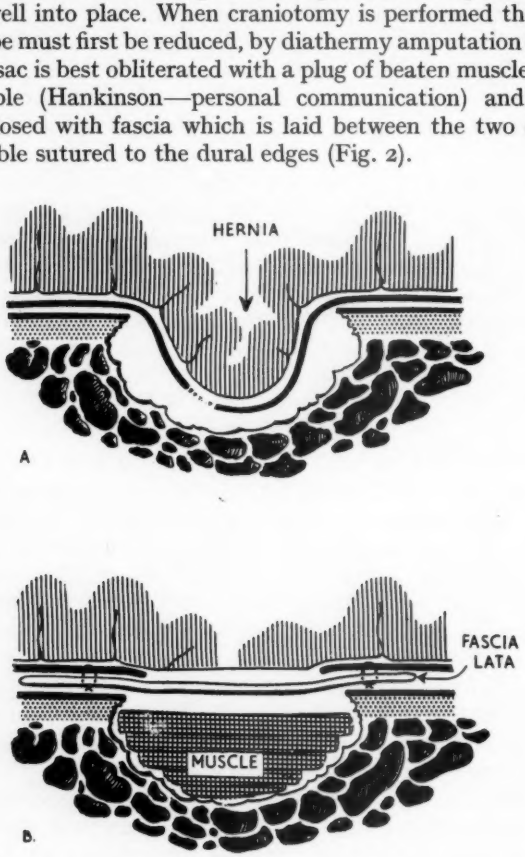

FIG. 2.
Showing (a) a cerebral hernia formed only by temporal lobe, and (b) the mode of repair.

Antibiotics should be given in all cases. Intramuscular penicillin and oral sulphonamides have been used and in no case has meningitis resulted from repair procedures, nor was evident meningitis made worse. The outlook in this condition has been completely altered by the advent of antibiotics. In the past patients with C.S.F. otorrhœa often died of meningitis; in this series all recovered despite the fact that in one case the leak was present for several months, antibiotics having cleared up intercurrent infections (Case IV). In no case has leakage recurred after treatment.

CASE I.

Mrs. E.D. was 33 years old when she was first seen. Five months previously the right side of her face began to twitch and after two months she developed a complete right sided lower motor neurone paralysis of the facial nerve. Because of radiological evidence of a cholesteatoma the the patient was referred on January 7th, 1960 to Mr. Munro Black, who confirmed the diagnosis of a right sided chronic suppurative otitis media. The patient denied ever having trouble in her right ear, even though directly questioned concerning aural discharge and giddiness. At operation on 15.6.60, the middle-ear cleft was found to be the seat of extensive cholesteatoma which completely filled the antrum and had destroyed the tegmen tympani leaving dura exposed. The labyrinth lay free as a sequestrum and cholesteatoma was found to extend as far as the petrous apex. After a careful removal of cholesteatoma a profuse flow of C.S.F. came from this region. This was partially controlled by a B.I.P.P. gauze wick which as left in situ. She was treated with Sulphamezathine and Penicillin. A C.S.F. leak persisted for several days. At theatre dressing on the third post-operative day (19.1.60) the cavity was repacked with B.I.P.P. The C.S.F. leak continued until the eighth post-operative day (23.1.60). On the tenth post-operative day the cavity was clean and dry. She was discharged home well on the nineteenth post-operative day. It was decided that it was not worth while trying to repair her facial nerve.

She has been seen regularly as an out-patient. Her mastoid cavity remains wet and infected although there is no C.S.F. leak.

CASE II.

Miss O.C. aged 19 attended in October 1950 with an acute exacerbation of chronic otitis media. A left radical mastoidectomy had been performed ten years previously. By May 1951 in spite of regular out-patient toilet the cavity remained unsatisfactory and she showed a positive fistula sign. A left labyrinthectomy with superior and inferior vestibulotomy was performed in January 1952. She continued to have vertigo which necessitated her admission to hospital in 1953 and 1954. The patient did not wish to have further surgical interference. It was only in 1959 that she could be persuaded to submit herself to further operation although during the last seven years and ten months a positive fistula sign could be elicited by mopping in the region of the round and oval windows.

The mastoid cavity was re-explored by Mr. J. D. K. Dawes on 24.10.59. The cavity, including numerous perilabyrinthine cells, was carefully cleared of granulation tissue. The superior vestibulotomy contained fibrous tissue but a new opening was made which entered the vestibule from above. The old inferior vestibulotomy had just cracked through the wall of the promontory and the opening was enlarged and carried through to the cochlea. Masses of granulation tissue were removed from

the vestibule and cochlea together with the remains of the membranous labyrinth. A profuse C.S.F. leak, presumably coming from the internal auditory meatus, appeared. The vestibule and cochlea were packed with temporalis muscle and the leak stopped. She made a good post-operative recovery but C.S.F. was seen soaking the dressing for three days post-operatively. B.I.P.P. packs were removed seven days after operation, and the patient discharged home. When she was last seen the cavity was healed, no fistula sign was present and she was symptom free.

Case III.

Mr. J. F. aged 58 was referred as an emergency on 24.4.56. He had been deaf in the right ear since 1918 after an artillery barrage. After an acute exacerbation of chronic otitis media ten weeks previously he complained of right sided frontal headaches, dizzy spells and difficulty in walking. He staggered and walked on a wide base. In the right ear there was a posterior attic erosion through which a cholesteatoma was visible. There was no fistula sign and no nystagmus.

During the next week he developed earache and was a little giddy. He was admitted to hospital three days later (4.5.56). Mr. J. D. K. Dawes performed a right endaural radical mastoidectomy on 5.5.56. A sclerotic mastoid was opened revealing granulation tissue and cholesteatoma in the antrum, aditus, attic, the middle portion of the sinus tympani and perilabyrinthine cells. The malleus was largely eroded, the incus had been destroyed and only the footplate of the stapes was found. The lateral sinus which was well forward was nicked with the drill whilst working in the sinodural angle. The opening was packed off and a postage stamp graft of temporalis muscle laid over it. The wound was partially grafted with skin and closed.

The night after operation a brisk C.S.F. leak developed. The next morning (6.5.56) the wound was re-opened postaurally and the packs and skin grafts removed. The leak ceased but recurred when the lateral sinus was displaced laterally, there being a dural defect in the sinodural angle. Two pieces of fascia lata were used, one being laid along the length of the sinus and tucked behind it medially, the other laid transversely to reinforce the repair to the posterior fossa dura and the wound was packed.

Penicillin was given for one week post-operatively and the patient made an uninterrupted recovery. After dressing the wound on the eighth post-operative day he was discharged home (15.5.56). He has remained well.

Case IV.

Mr. J.W. was aged 24 when he was admitted as an emergency on 22.11.55. He had had right otorrhœa since childhood and left otorrhœa

for three years. Following a blow on the right side of the head a week previously the otorrhœa increased and he developed progressively more severe earache in the left ear. He had postural vertigo, had vomited for three days, had rigors for two days and severe occipital headache with right sided neckache for one day. He had neck stiffness, a puffy right eyelid and a positive Kernig's sign. When fœtid pus was cleaned from the right ear the attic was seen to be packed with cholesteatoma. He was tender over the right jugular vein.

A diagnosis was made of otogenic meningitis, right lateral sinus thrombosis with extension to the jugular vein, and a probable cavernous sinus thrombosis. Lumbar puncture showed the C.S.F. pressure to be over 300 mm. and the cell count 30/c.mm. Treatment was instituted with intrathecal penicillin and oral sulphamezathine.

Next day he was much worse and Mr. McGuckin explored the right ear postaurally. A hard cortex was opened releasing pus under pressure from the mastoid cavity. There was an osteitis of the tegmen tympani leading to a thrombosed superior petrosal sinus. The dura over the lateral sinus was grey-green and the region of the genu was black, the result of clot inside the vein, which was incised, black pus and clot being removed from the sinus which was then packed with B.I.P.P. gauze. There was a small subdural abscess just anterior to the superior petrosal sinus. A mass of cholesteatoma was sucked out of the middle-ear cleft, the cavity was packed with B.I.P.P. gauze and the wound left open.

Following operation he made a good recovery but two days later he had further rigors and severe pain over the right jugular vein. Treatment with anticoagulants was commenced, and continued for eleven days. He gradually improved and four weeks after the original operation (21.12.55) Mr. McGuckin completed the right radical mastoidectomy. The remains of cholesteatoma were sucked from the attic and aditus and almost the whole of the sinodural angle was drilled out to clear cells filled with pyogenic membrane. Making a satisfactory recovery the patient was discharged home nine days later (30.12.55).

At follow up three months after discharge from hospital the deep part of the cavity was grossly stenosed. He failed to attend for eighteen months and when seen again on 20.12.57 he complained of severe right temporal pain and a profuse watery otorrhœa. This recurred quickly after mopping. He was admitted and treated by local toilet and systemic penicillin but was discharged home after two weeks much improved.

Three months later (31.3.58) he was again admitted with headache and vomiting of three days duration. The clear right sided otorrhœa was still present but ceased after systemic penicillin and streptomycin therapy. At follow up the right otorrhœa was again noted, sometimes consisting of mucopus, sometimes it was clear. The cavity was grossly stenosed, by a prolapse of posterior fossa dura. Six months later (24.10.58) he was again

admitted because he had vomited for two days. Mr. J. D. K. Dawes decided that the clear otorrhœa was a C.S.F. leak and a revision of the right radical mastoidectomy was performed on 26.10.58, three years after the original operation, and ten months after the clear discharge had first been seen.

The meatal stenosis was caused by a false membrane which covered granulation tissue and dura of the posterior fossa, this latter had herniated into and obliterated the mastoid cavity, bulging forward into the external auditory canal. A large cholesteatoma which was removed was present between the stenosis and the medial tympanic wall. The posterior fossa dura was readily displaced backwards, revealing deep to the bone of the posterior fossa, a dural herniation which was closed with a strip of fascia lata.

The patient made an uneventful post-operative recovery and after a theatre dressing was discharged home on the twelfth post-operative day (8.11.58).

He has been seen regularly since then as an out-patient. His right ear is clean and dry although within four months of the last operation his cavity was again stenosed. He remains well.

Case V.

L.F. was seven years old when she was admitted to the Royal Victoria Infirmary under the care of Mr. McGuckin on 6.8.55.

Fifteen days previously she developed a febrile illness and severe right sided earache. On the fifth day of her illness she was admitted to a pediatric unit with meningitis and purpuric spots on the trunk. She had previously had two operations for Fallot's tetrad. After the second operation a left hemiplegia developed which had partially resolved.

In spite of the history of earache her ear was not examined and on clinical grounds only the meningitis was considered to be of meningococcal type. She was treated with systemic and intrathecal penicillin and sulphadiazine. She improved but slightly on this regimen and on the fourteenth day of her illness, nine days after admission to hospital, she developed a profuse right sided cerebrospinal fluid otorrhœa and a temperature of 105°F. Next day she was seen by Mr. McGuckin who immediately transferred her to the Royal Victoria Infirmary.

Apart from the obvious meningitis a history was obtained of a long standing right sided otorrhœa which had been fœtid for two years. On examination she was ill, had a marked cyanosis of lips, cheeks, fingers and toes and her right ear was pouring C.S.F. and deep in the external auditory canal masses of cholesteatoma were seen. Later the same day Mr. J. D. K. Dawes explored her right ear.

A postaural approach was used. Cholesteatoma filled the whole of the mastoid cavity except for a few tip cells, and extended forwards from

the antrum to the attic. C.S.F. was pouring from a perisinus abscess cavity. The cholesteatoma was cleared revealing an intact tegmen tympani but the lateral sinus plate was deficient over an area of about 1 sq. cm. The lateral sinus was surgically exposed from upper to the lower genu and as far forward as the jugular bulb. C.S.F. was pulsating from a posterior fossa dural defect, 3 × 2 mm. in size, which lay about the level of the internal auditory meatus, closely related to the lateral semicircular canal. The area was packed with gelfoam and B.I.P.P. packs.

Her condition improved post-operative. By the third post-operative day (9.8.55) the C.S.F. otorrhœa had disappeared and the Kernig's sign and neck stiffness had almost gone, but on the fifth post-operative day the C.S.F. leak reappeared and by the next day had become profuse. The packing and gelfoam were removed from the mastoid cavity and a small piece of amnion placed intradurally through the C.S.F. fistula. Bleeding from the lateral sinus occurred and was controlled by a temporalis muscle graft. Fascia lata was laid over the dural deficiency deep to the bony erosion at the site of the C.S.F. fistula and packed in position with gelfoam. Thereafter she made an uninterrupted recovery and the radical mastoidectomy was completed on 31.8.55. The child was discharged home fit and well, fifty four days after the onset of her illness (12.9.53). She was seen six nonths later when her cavity was clean and healed.

Case VI.

Mrs. I.H. aged 23 had a left radical mastoidectomy performed in 1940 after which she was trouble free for fourteen years. When seen again on September 7th, 1954, she gave a three weeks history of left sided otorrhœa. A large red mass presented in the posterior wall of the external auditory canal which, at suction clearance performed two weeks later (23.9.54), was found to be thickened skin. The skin was incised and pus with sodden cholesteatoma was seen in the cavity beyond. On 29.9.54 a formal endaural revision of the mastoidectomy was performed. The thick posterior wall swelling consisted of a mass of fibrous tissue covered by skin more that ½ cm. thick, attached directly to a forward bulging lateral sinus. The bony roof, the sinodural angle and posterior wall of the mastoid antrum had been removed at the previous operation. Dura of the middle and posterior cranial fossae had prolapsed into and filled the mastoid cavity pushing the lateral sinus against the anterior meatal wall. No attempt could be made to deal with the tympanic disease, but debris was sucked out of the cavity as carefully as possible. Skin grafts were used to cover the lateral sinus in an attempt to form a channel for the external auditory meatus and the upper end of the wound was closed with a flap of temporalis muscle, the wound being packed with B.I.P.P. gauze.

She was followed regularly as an out-patient. In spite of a B.I.P.P. pack left in situ for several weeks, the canal narrowed and by January 1956, sixteen months after operation, the dura was again bulging markedly into the deep part of the cavity.

All was well until 9.5.58, three years and eight months after operation, when a thin watery discharge was seen in the ear. It was C.S.F. She was admitted at once and treated with sulphadiazine and systemic penicillin.

There was never any evidence of meningitis, nor were any neurological abnormalities detected. She was seen by a neurosurgical colleague, Mr. J. Hankinson, who agreed to undertake the repair of the dural defect which he did six weeks after the onset of the C.S.F. otorrhœa (19.6.58).

At operation the floor of the left middle fossa was exposed through a formal temporal craniotomy. On elevating the temporal lobe a 1½ cm. defect was seen in the petrous bone, through which a tongue of brain tissue passed. This was excised, the resultant bony cavity was filled with a plug of beaten muscle and dural defect closed with a double layer of fascia lata sutured into position.

She made an uninterrupted recovery and remains well. Her cavity is dry and no further dural prolapse has been seen.

Summary and Conclusions

Cerebrospinal fluid otorrhœa arises in chronic destructive otitis media when the surgeon or the disease has opened some part of the subarachnoid space not already walled off by inflammatory barriers or plugged by herniating brain. Six cases are reported. Those cases in which the lesion was consequent on surgical trauma recovered with packing of the wound, temporalis muscle providing suitable material for packing. Chronic leaks developed when dural defects were kept open by the secretory pressure or by inflammatory adhesions. These required repair. Fascia is a suitable material for the repair of dural defects. For defects in the middle cranial fossa craniotomy gives the best approach whilst the transmastoid route is more suitable in the posterior fossa. The dangers and the advantages of antibiotics in this condition are stressed. All cases made a satisfactory recovery and there have been no recurrences of C.S.F. otorrhœa.

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TRACHEOSTOMY AND CONTROLLED RESPIRATION*

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THE contribution which I, as a Laryngologist, can offer to this discussion on tracheostomy and controlled respiration, is based on the following experience. During the period November 1956 until December 1959, my colleague, Dr. Russell Campbell and myself performed 224 tracheostomies. The majority of patients submitted to operation had head injuries—admitted to Newcastle Regional Neuro-surgical unit—which serves an area of heavy industry. Respiratory insufficiency is our problem, and although we see it most frequently in head injuries, we attempt to treat it as it occurs in cerebrovascular disease, and in acute or chronic chest infections. Cases of chest injury, tetanus and drug overdosage are also included in the total. All the patients were treated in the Newcastle General Hospital. We have had no experience with poliomyelitis.

Initially, the cases submitted to tracheostomy were severe brain-stem injuries, in an attempt to lower the very high mortality prevalent in this type of trauma. Eighty-seven cases were seen on admission by the same neurosurgeon, anæsthetist and myself. This experience provided the basis for the regime which is now in use. The number of patients requiring controlled respiration was not high, but the application presented no difficulty.

I now give some of our conclusions:

Respiratory insufficiency may be defined as a condition in which the amount of oxygen and carbon dioxide in the blood stream is altered by an abnormality in the respiratory system. Any unconscious patient may provide some degree of insufficiency—and in particular I would refer to the head injuries which have formed the bulk of the material. *Anoxia* appears to be the main cause of death in patients who survive the initial accident. Previously, tracheostomy had been advocated as a satisfactory method of dealing with chest complication *at a late stage.* We feel that respiratory insufficiency exists almost from the beginning in the unconscious patient, and if it is corrected early, pulmonary complications may be avoided, and mortality reduced.

Clinical information available prior to 1956.

In common with other workers, we have observed the following features in patients suffering from severe head injuries. At post-mortem

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many cases showed vomit and blood clot in bronchi and bronchioles, together with histological evidence of destruction of respiratory epithelium, even when there had been *no clinical evidence of chest infection before death*. Sub-endocardial hæmorrhages were seen over the bundle of His, suggesting a significant degree of anoxia. These observations led us to consider earlier aspiration of secretions through a endotracheal tube, and a marked improvement frequently resulted. Such improvement was not maintained if the tube was removed.

Control of anoxia required that we give attention not only to pathology in the chest, but *also* to pathological changes in the brain-stem. In severe damage to the pons, medulla, and hypothalamic areas, the respiratory centre may be seriously depressed by direct injury, or by vascular impairment. Interference with the vagal centres, as in bulbar poliomyelitis, may produce loss of control in the pharynx and larynx, with subsequent overspill into the lung-tree. Moreover, hypothalamic trauma may produce a sympathetic-parasympathetic imbalance, leading to disastrous pulmonary œdema.

Tracheostomy is of course directed mainly at the condition of the chest. We have to consider the unsuspected presence of inhaled gastric contents, occurring usually at the time of the accident when the vomiting centre is stimulated. We may be dealing with a chest which was not fully efficient prior to the accident, especially in older people, in whom chronic bronchitis is common in our area. Incorrect positioning of the patient, especially during transportation, is a hazard which is often overlooked. Lingual obstruction when lying on the back, and blood or cerebrospinal fluid passing through an incompetent larynx, lessen the patient's chances of survival. If all these factors are treated quickly and adequately, then even the most severely injured patient may survive without handicap.

The question of how much harm may be done to patients by submitting them to tracheostomy must be answered. Tracheostomy of course, always carries a risk of superadded infection in a chest. This risk must be accepted at times, and especially in those patients who, because of injury, disease, or overdose of drugs, require controlled respiration. Nevertheless, every effort must be made to reduce, or eliminate secondary infection. Our patients have to be treated in open wards, and are, of course, subject to cross infection. There may be ten or even more patients in varying stages of treatment at the same time. We are sure that the use of cubicles and strict attention to asepsis would prove beneficial. Efficient nursing is an invaluable aid.

Now as to the various measures we tried in an attempt to overcome anoxia. The neurosurgeons attempt to control brain-stem damage by the use of varying strengths of plasma, mixtures of drugs such as Largactil, Pethilorfan, and Phenergan, and certain operative procedures at the discretion of the neurosurgeon. Treatment of the chest pathology

requires adequate oxygen supply, and elimination of infection, or foreign materials, to allow of full oxygen-exchange, and prevent further brain-stem damage by continuing anoxia. We found the use of a simple oxygen face-mask, and antibiotic cover inadequate. Patients still died, and we found post-mortem evidence of anoxia, with severe lung damage. The oral airway and pharyngeal suction provided no solution. Next, we used cuffed endotracheal tubes, but many patients would not tolerate them, and their long term use led to permanent laryngeal damage. This measure, is however, useful in an emergency situation. In our opinion, endo-tracheal tubes should not be retained for longer than 12 hours.

It now became necessary to consider tracheostomy as an effective means of maintenance of the airway, giving ease of suction, and reduction of respiratory dead-space, by by-passing the mouth, pharynx and upper trachea. To begin with, we limited ourselves to patients who had obvious clinical or radiological signs of infection or foreign material in the chest, or who had severe facial injuries with blood and cerebrospinal fluid in the pharynx. The results were not satisfactory. Despite free oxygen supply, aspiration of bronchi, and antibiotic cover, we continued to lose patients, and found the same post-mortem signs of anoxia, and even more extensive chest pathology than we had suspected. Therefore, we extended the indications to include all deeply unconscious patients, and we assumed that their chests might well contain noxious material. The benefits of this extension were immediately apparent, since the majority of the patients had indeed a lot of secretion in their chests, and they improved as soon as the tracheostomy regime was established, the chest sucked clear, and oxygen administered. From then on, the mortality rapidly fell, and the fall continues to date. *In severe brain-stem injuries the mortality has been reduced from 80 per cent. to just above 30 per cent.* It is now our policy to employ tracheostomy in all deeply unconscious patients who do not show rapid return to consciousness. In any doubtful case, an endo-tracheal tube is passed, and may be left in place for 12 hours. If the patient has not then recovered, and if the cough reflex is inadequate, tracheostomy is undertaken. Unconscious patients have an œsophageal tube passed on admission and aspiration continued until recovery is complete.

Children have not proved such a difficult problem, and rarely have we had to perform tracheostomy. It appears quite easy to control their chests by positioning, supplemented by antibiotics and physiotherapy. However, it should still be regarded as possibly a life-saving measure in children with respiratory insufficiency.

As regards the operation itself we use generally accepted techniques. A curved tranverse incision is used, and general anæsthesia is the rule. The trachea is exposed just far enough to allow the largest opening to be made. The minimum of dissection reduces the incidence of surgical emphysema. The question of high or low tracheostomy is decided on

individual cases. When there is troublesome over-spill of secretions, or when controlled respiration is required, a high tracheostomy, with division of the thyroid isthmus is employed—otherwise there may be difficulty in positioning the longer cuffed tube, which will tend to touch the carina, or even enter the right main bronchus. In cases with little or no overflow, and not requiring controlled respiration, a low tracheostomy is employed. We have experienced troublesome bleeding in patients having triple plasma. The production of a dry field before opening the trachea is often time-consuming, but clearly essential. We have tried all types of tubes, and now use Durham Lobster Tail for cases with no overspill. If the trachea is to be totally occluded, as in controlled respiration, or for control of secretions from above, a specially shortened cuffed McGill endotracheal tube is used. The tracheostomy is nowadays performed before any surgical intervention, since anæsthetists consider the patient's condition to be more satisfactory for any further measures.

The purpose of the tracheostomy is to provide a constant clear airway. The diameter of the tracheostomy tube is *not* equivalent to that of the trachea, and therefore, any obstruction, even temporary, may be harmful. Subsequent management is mainly carried out by nurses, and special training and education is essential. *Asepsis* is important, for example, suction catheters are kept under cover in antiseptic solution, and bed clothes are covered with towels before suction is performed. We do not think that surgical gloves are essential, and we rely on scrubbing of hands and change of gowns between patients. Neglect of these precautions may result in local infection of the chest, thickening of secretion and crust formation, with possible obstruction to the tube. A small accumulation of secretion in the tracheostomy tube markedly increases both resistance and oxygen-lack, perhaps to a dangerous level—especially if a cuffed tube is used on controlled respiration. The problem of adequate suction is still not completely solved. In common with others we have found that the left lung is more at risk. Despite positioning, and the use of angled-catheters, it is extremely difficult to enter the left bronchus, a fact which may be verified radiologically. Patients dying from chest infection usually show massive infection on the left side, and those dying from other causes commonly show the left lung to be much worse than the right. Constant attention to the left lung is essential, and any persistent block must be dealt with by bronchoscopy. We find that the left lung is only emptied at the cost of overflow into the right side, and is constantly presenting us with new problems.

We have found that bronchoscopy is not without risk and its use is limited to a rapid operation for removal of a persistent block in a main bronchus. Despite adequate pre-medication with atropine, there have been several cases of cardiac arrest during bronchoscopy, and one proved fatal.

The tube must be of the largest possible size, and the inner tube should be changed frequently, especially during the first 48 hours. During this early period crust formation is at its maximum; partly because of exposure of the tracheal and bronchial mucosa to the air, and partly because many of the head injuries are intentionally dehydrated in the early stages of treatment. Proper humidification of ingoing oxygen is the most important means of keeping lung secretions moist, and thus easily removable. Many patients progress very well throughout the period of tracheostomy without the need for other means of control of secretions. Tenacious sputum may be particularly troublesome after inhalation of gastric contents, and we have found certain preparations worth trial:

Alevair—which is a type of detergent, has a limited use, and is not very effective against mucoid sputum.

Trypur—which is a pure crystalline trypsin is most effective against any inhaled food particles, and we have little doubt of its efficiency in breaking them up, so as to render them accessible to suction. Both preparations are instilled into the lungs by a Collinson's inhaler.

Parenzyme—is also a trypsin, which can be given intramuscularly, and for this reason we use it in preference to the others. We have not had any adverse side effects. All patients are given intramuscular penicillin and sulphadiazine. If chest infection has occurred, and is not thus controlled, we use intramuscular terramycin. Routine bacteriological investigation has not proved necessary. In addition 1,000,000 units of aqueous penicillin are instilled into the lungs daily *viâ* a Collinson's inhaler.

We require a chest X-ray on return from the theatre, for the following reasons:

1. To show the position of the tracheostomy tube, especially its proximity to the carina, and the main bronchi.

2. To demonstrate the presence of any areas of collapse or collections of foreign material.

Physiotherapy should be employed early, since it is probably the most effective way of keeping the left lung clear. I am sometimes surprised how much material can be moved up and eventually sucked clear by efficient physiotherapy.

Intravenous fluid is used until an adequate cough reflex returns. Feeding *viâ* the œsophageal tube can then be started, and the tracheostomy wound allowed to close. Fortunately we have not had any complications following tracheostomy. From our experience we would summarize the indications for, and advantages of tracheostomy in patients with respiratory insufficiency as follows:

1. In all patients who require controlled respiration for any length of time.

2. In the deeply unconscious patient—as soon as possible. Prophylactically to prevent aspiration into the chest, and where aspiration is

liable to have occurred, although there may be little evidence of this on clinical examination.

3. In the semiconscious patient where recovery is stationary. Again prophylactically against aspiration and because we believe many to be suffering from hypoxia and hypercapnœa—which produce headache, restlessness, and disorientation, and, at a later stage, respiratory depression and circulatory collapse.

The advantages are:

1. Allows aspiration and control of secretions.

2. Oxygen can be administered at correct temperatures and humidification.

3. It by-passes an incompetent larynx and pharynx.

4. It by-passes the respiratory dead space in the mouth, pharynx and upper trachea, thus making a somewhat inadequate respiratory effort more effective.

5. It allows easy change over to controlled respiration.

Finally, we have always stressed that tracheostomy for head injuries should be done as soon as the indications are present, and we now have many patients transferred having had a tracheostomy performed in the hospital to which they were admitted. My neurosurgical colleagues are satisfied that the prognosis of these patients is improved.

During the winter the physicians admit patients with acute on chronic chest infections. Many of these cases have thick lung secretions, and a poor cough reflex. Tracheostomy, with suction and oxygen, changes these patients from choking cyanosis to a state in which they can survive until antibiotics have controlled the infection. We are asked to see an increasing number of these cases, and we hope to be able to comment at greater length as our experience increases. A few cases of congestive cardiac failure have had tracheostomies to control the pulmonary œdema while the requisite drugs take effect on the cardiac condition.

Patients with tracheostomies require a lot of medical and nursing care; the application of controlled respiration means constant supervision—our opinion is that a separate ward or unit is the only means of maintaining such supervision—we have found it quite impossible to cope with such patients being treated in various wards. Should we ever obtain such a unit, I am certain the care of our tracheostomies would be much improved.

I wish to thank my neurosurgical and medical colleagues for allowing me to use their cases for this paper, also my anæsthetic colleagues who play a large part in the care of the patients, and the Nursing Staff on whom so much depends.

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TRACHEOSTOMY AND CONTROLLED RESPIRATION*

By E. A. COOPER (Newcastle-upon-Tyne)

THE two fundamental necessities for controlled respiration are a controlled airway and a controlled, alternating pressure. Mr. Frew has spoken from his own personal experience of head injuries, and has emphasized the importance of keeping a clear airway, free from obstruction by the tongue and pharyngeal contents. He has also dwelt on the need for bronchial toilet. But almost all his patients were capable of providing their own alternating pressure. I wish to discuss patients who are not.

We, as anæsthetists, are called to many respiratory emergencies but only a minority of the patients need artificial respiration. In general, we would not advise tracheostomy unless we expected the patient to be under our care for more than 24 hours. Thus the title of the paper "Tracheostomy and controlled Respiration" defines the clinical material under discussion as patients who need artificial respiration for more than 24 hours. In our recent experience such patients have numbered about 8 per year. Review suggests that about 3 of these 8 are subsequently shown not to have been saveable from the outset, owing to the severity of their disease. Of the 5 potential survivors, about 3 go home more or less fit, 2 do not.

The mechanism of the respiratory failure falls into one of 3 groups —medullary, neuromuscular, and mechanical. In my own experience the following conditions have occurred,

(a) medullary failure,
overdosage with drugs,
cerebral vascular accidents,
cerebral tumours, especially with hæmorrhage,
anoxia following circulatory arrest,
head injury,
various inflammatory processes such as encephalomyelitis and meningitis with vascular thrombosis,

(b) neuromuscular disorders, working anatomically down the track,
tranverse myelitis,
poliomyelitis,
"acute polyneuritis" in its various guises,

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myasthenia gravis,
the treatment of tetanus by neuromuscular blocking agents,
collagen diseases such as polymyositis and scleroderma,

(c) mechanical troubles,
chest injuries in which multiple fractures have led to paradoxical movement and flail chest,
the disorders of pulmonary mechanics which cause respiratory failure in emphysema.

But whatever the disease, all these patients can be classified that they either have or have not, adequate control of their airway. On this we decide what type of respirator is appropriate. The "cabinet" or "iron lung" sucks air from the pharynx into the lungs. If the patient cannot control his pharynx and larynx, the cabinet can then also suck saliva or soup or vomit into the lungs. We think that inability to breathe, together with inability to control the airway, necessitates the use of a tracheostomy with a cuffed tube. With this we would, in general, use an intermittent positive pressure respirator, blowing the air direct into the trachea. Indeed, unless there was a strong contra-indication to tracheostomy, we would treat almost all our long-term respiratory failures in this way. I therefore wish to discuss, from a general and fundamental point of view, the tracheostomy and what is required of it, in patients who need prolonged artificial respiration, speaking primarily of those who are treated by an intermittent positive pressure respirator.

We require of the tracheostomy that it provide a guaranteed airway, through which the respirator can be guaranteed to work, in such a way that the patient can be guaranteed the best chance of survival. Almost all considerations, clinical, experimental and theoretical, show that these needs can most easily be met by making a big hole in the trachea and using a big tube. A guaranteed airway requires a tube which will not block and can be cleaned easily, that is, a big tube. The airway also includes the lungs, and since the patient cannot cough, bronchial suction is needed. Thus a suction catheter of reasonable bore must be easily manœuverable, which again necessitates a big tracheostomy tube.

The tube is also the adaptor between the respirator and the lungs. In these patients it is not an alternative airway, not a mere convenient by-pass, not just a useful point of access to the bronchial tree. It is the only airway and the only means of applying the alternating pressure. If the tube comes out, the patient may be unconscious from anoxia in 90 seconds and dead in another 90. It is essential to have, as nearly as possible, guaranteed fixation. In our experience the standard methods are satisfactory for adults, but we believe that in children the flange of the tube should be stitched to the skin. Even so, absolute fixation cannot be certain. Everything reasonable must therefore be done to assist at the moment of crisis should the tube slip out. Since this is a 24-hour

risk it is unlikely that a person of high skill will be available within a few seconds. It is therefore essential that a window be cut from the trachea and that it be big.

The requirements of the respirator will now be considered. A normal patient, at rest in bed, has a fairly constant respiratory activity with a minute volume of the order of 4-8 l./min. When he is on the respirator he again requires a fairly constant minute volume, but one which is considerably bigger. This is for three chief reasons. First, the respirator works best in a relaxed patient, not on one making his own respiratory efforts. These efforts are controlled by the tension of carbon dioxide in the arterial blood. Consequently, if it is desired to control the patient's respiration, it is necessary to keep the arterial CO_2 tension below the normal critical level which initiates respiration. This requires hyperventilation. Second, it seems that artificial respiration is probably less efficient than normal breathing for the elimination of CO_2 from the lungs. This means that more hyperventilation is necessary to maintain low arterial tensions of CO_2. Third, the patient's production of CO_2 is likely to be increased by pyrexia and anxiety. Thus, even more ventilation is needed. In general, it is desirable to be able to provide ventilation two or three times as much as the normal resting, up to 15 l./min., and to maintain it fairly constant. For this, most respirators require a tracheostomy tube of constant, low resistance. The general laws governing the resistance of tubes will therefore be discussed.

It is desired to pass air at relatively high rates of flow, at least double the normal. But, for tracheostomy tubes, double the rate of flow requires more than double the pressure gradient. Pressure and rate of flow are not related by simple proportionality but by a square law. Thus,

TABLE I.

Tube	Ratio of pressure necessary to maintain rate of flow of 20 and 10 l./min.
Negus No. 28	3·7
Negus No. 32	3·5
Negus No. 28 partially occluded	3·6
Negus No. 32 partially occluded	3·8
James No. 8	3·5
Radcliffe No. 8	4·0
Plain Rubber No. 8	4·4
	Mean .. 3·8

Pressure gradient $\propto$ (rate of flow)^2, and a doubled rate of flow can only be maintained by a pressure increased four times. Table I confirms this theoretical prediction. The results of experiments carried out on tracheostomy tubes show that when the flow of gas is raised from 10 to

20 l./min., the pressure necessary to provide the flow is increased almost four times.

The relationship between the pressure and the bore or radius of the tube is more complicated. Thus,

Pressure gradient $\propto (\frac{1}{\text{radius}})^5$. Hence, if flow is to be maintained through two tubes one of whose radius is half that of the other, the pressures necessary will be in the ratio 2^5:1, that is 32:1. This relationship has also been confirmed experimentally. For instance, the internal diameters at the outlet of Negus tubes, numbers 28 and 32, were found to be 7 and 8 mm. The pressures necessary to maintain the same rates of gas flow would be expected to be in the ratio $(\frac{8}{7})^5$, which is 1·95. In experiment, the ratio was 1·88. It is reasonable therefore to enquire into the effect on resistance of secretions lodged in the tube. For theoretical considerations a reasonable volume of secretions can be taken to be 1/10 ml. If an equivalent area of a Negus 32 tube were obstructed, its resistance would be increased to three times normal. But the same volume of secretions would occupy a much higher proportion of the lumen of a No. 28 tube. Indeed, the resistance of a No. 28, containing the same 1/10 ml., of sputum would be increased to eight times its normal.

The clinician must always ask the theoretician whether his theories matter in practice. The following results show that they do. The inner tube (Negus No. 32) was taken on 10 separate occasions from a patient recovering from severe chest injuries. The times at which it was taken were chance throughout a number of days. Sometimes the patient was on the respirator, sometimes breathing spontaneously. The resistance of the tube to gas flow was measured in quadruplicate on each occasion and the mean taken. The resistance of the tube at the same rate of gas flow when absolutely clean had previously been measured. Taking this to be 100 per cent., the resistance of the tube when dirty was also expressed as a percentage. The results are shown in Fig. 1. On 6 occasions the resistance was not much greater than normal (100 per cent.). But on 3 occasions it was more than double and on a fourth it was increased fourfold. Fig. 2 is a photograph of the end of the tube on the occasion of the tenth test. It will be seen that very little occlusion by secretions had sufficed to double the resistance. Indeed, measurement showed that the loss of cross-sectional area was a little over 10 per cent., which agreed very well with the foregoing mathematical theory. It is instructive to consider the relative resistances of tubes of different diameters with and without obstruction by this same volume of secretions. Examples are shown in Table II, expressed as percentage of the resistance of the clean No. 32 Negus. It is shown that if a tube of the same shape existed whose diameter were 10 mm. (one quarter as large again as the 32) its resistance when dirty would still only be one half of that of the 32 when clean.

These considerations have all been of relative resistance and it is

necessary to relate them to the actual additional pressure against which the respirator may have to work. At the highest flow rate likely to be required, 217 per cent. of the resistance of a No. 32 Negus tube represents an added pressure gradient of about 10 cm. H_2O in a system in which normally about 20 cm. H_2O would be used.

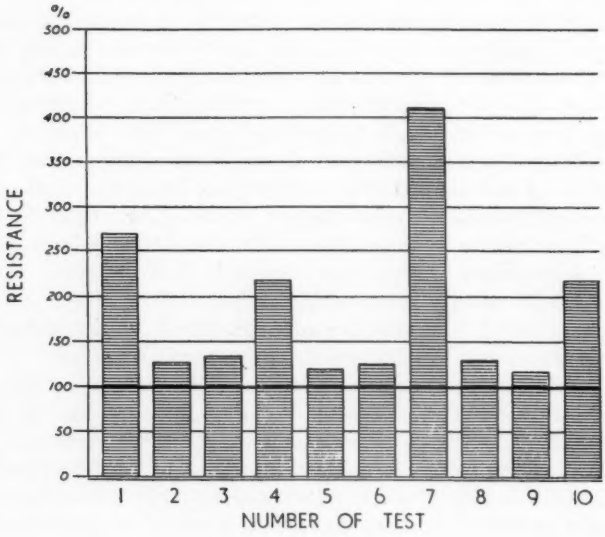

FIG. 1.

The resistance to airflow of a tracheostomy tube (inner No. 32 Negus) when taken direct from clinical use, as compared with that of the same tube when absolutely clean (100%).

Having considered the airway at some length, it is necessary to review the other function of the tube, that of adaptor between the respirator and the patient's trachea. The space between the tube and the tracheal wall must be filled. If it is not, air directed towards the lungs may leak away into the pharynx. Also, if there is a space between the tube and trachea, the pharyngeal contents may contaminate the lungs. The space must be filled by the cuff and the bigger the space the greater the burden. Rupture of the cuff may be fatal. The space should therefore be as small as practicable, and this implies, yet again, as wide a tube as possible. Indeed, in clinical practice, we have only been free from troubles with cuffs when we have used big tubes.

So far, the respiratory system alone has been discussed. I would now like to consider briefly the tracheostomy in relation to the circulation. The normal, healthy, breathing patient generates his own alternating pressure. Intermittently, he makes his intrathoracic pressure negative

and air is drawn through the trachea into the chest. Similarly, blood is drawn into the chest through the great veins. This mechanism is a major feature in the maintenance of venous return to the heart, and hence of the circulation. In the patients we are considering, there is no inspiratory effort and no suction into the heart. Indeed, inspiration is replaced by

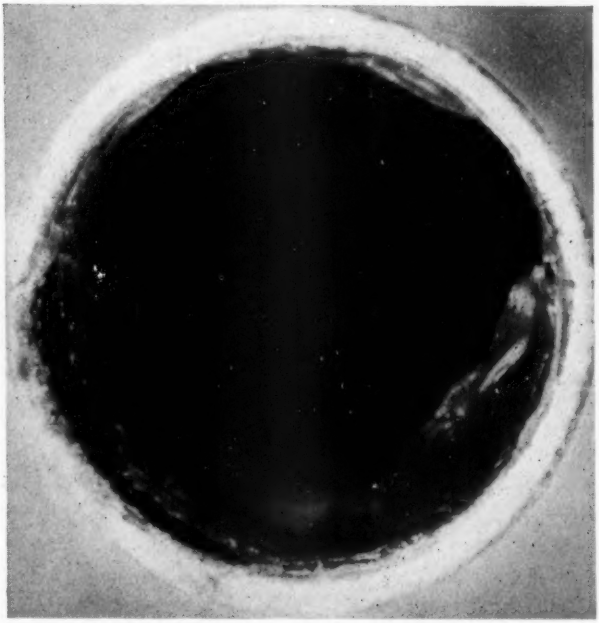

FIG. 2.

Photograph of the outlet of same tracheostomy tube (inner No. 32 Negus) at the time of test No. 10 of Fig. 1, showing the amount of secretions which had sufficed to more than double the resistance.

TABLE II

Tube	Diameter of outlet orifice in mm.	Resistance to gas flow expressed as % of that of No. 32 tube when clean	
		Clean	Lumen obstructed by same mass of secretions as in Fig. 2
Negus inner No. 28	7	195	573
Negus inner No. 32	8	100	217
Hypothetical Negus inner having outlet orifice=	10 mm.	33	52

a phase of inflation under positive pressure which tends to dam back the return of venous blood to the heart. The circulation is best served by a short application of positive pressure by the respirator, and also by the allowance of easy expiration so that the air flows out fast and the pressure inside the chest falls quickly. The need for rapid inflow of air and for easy, quick expiration once again emphasizes the need for a tube of reliable low resistance, that is, a big tube. In the patient whose circulatory system is in good condition, the size of tube is not critical. But in someone with severe toxæmia and dehydration, circulatory embarrassment may arise from mal-application of intermittent positive pressure. Some clinicians favour the use of a respirator which itself provides a negative pressure during expiration.

This paper has been an eulogy of the wide-bore tube and it would be wrong to end without considering the possible disadvantages. First, it may be technically more difficult to remove a large window from the trachea and introduce a large tube. Second, a large hole gives less secure fixation of the tube. Third, a large tube may in theory increase the risk of pressure damage to the tracheal wall but this is unlikely when the tube is not actually a tight fit in the lumen. Fourth, the larger hole may carry a greater risk of subsequent non-closure or of closure with stenosis of the trachea. I do not think that I have seen any of these events to a significant degree.

To summarize, I have been speaking of a small number of patients who are, medically, a rather esoteric group. Most of them are very ill but many can usefully be saved. I believe that the biggest number can be saved by the biggest hole and the biggest tube.

Acknowledgement

I am very grateful to Professor E. A. Pask for many helpful suggestions during the preparation of this paper.

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TRACHEOTOMY AND CONTROLLED RESPIRATION*

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The Mechanical Aspect and Treatment

Tracheotomy having been established it is possible that respiratory power may gradually or suddenly fail.

When ventilation becomes inadequate there should be no hesitation in seeking mechanical aid. The vicious spiral of anoxæmia and pulmonary œdema is all too easily initiated.

With progressive conditions a vital capacity below one litre usually calls for aid.

In the tracheotomized patient it is far easier to employ positive pressure than a Cabinet Respirator.

(1) Access to the patient for every purpose is facilitated.

(2) The problems of avoiding the tracheotomy with the neck seal are obviated.

(3) Humidification is easy.

(4) The patient is less constrained.

(5) The necessary apparatus is less bulky and easier to store.

As regards machinery the essential is complete reliability which includes simplicity both for servicing and easier understanding by the Nursing Staff. If possible uniform apparatus should be employed throughout the department.

Machines

Generalizing—machines are of two main types.

(1) "Volume Cycled" where a fixed charge is delivered at each stroke.

(2) "Pressure Cycled" where the patient is filled to a pre-determined pressure which initiates the expiratory phase.

"Volume Cycled" machines are mainly electrically driven pumps utilizing room air.

"Pressure Cycled" employ air previously compressed either in cylinders or by separate generator.

The most popular machines in this country have proved to be volume cycled—possibly wrongly. This is probably because:

(1) The machines are easier to make and understand and their faults

* Paper read at Section of Laryngology, Royal Society of Medicine, February 5th, 1960.

are immediately obvious. Accordingly they were turned out rapidly in the early, rather amateur days.

(2) Controls are simple and separate whereas in the other types they are generally over-sensitive and tend to interact.

(3) The Pressure Cycled machines have the serious danger that with a blockage the pressure will rise rapidly and the cycle will occur, but the patient will receive precisely nothing.

(4) Cylinders of compressed air are bulky and the number used is alarming.

Whatever the machine certain values obtain:

(1) A speed of about 17 r.p.m. is almost standard for adults, in special cases with diminished available alveolar space, about 22 r.p.m. is employed and maybe 26 r.p.m. in children.

Beyond these speeds serious problems of airflow arise.

(2) About 12 cms. water pressure is normally needed to maintain inflation of all parts of the lung, and over 25 cms. will lead to almost certain emphysema.

12-25 cms. is therefore the pressure range.

(3) Inspiratory—Expiratory phasing is around the 1 to 2 ratio—not shorter and the rise of flow rate (pressure curve) should be rapid but not excessively so. This may lead to over-distention of proximal alveoli while starving the periphery, owing to bronchial tree resistance at excessive flow.

(4) Hyper-ventilation is inevitable and seems to do no harm.

In our experience at this moment a child of six required 5 litres per minute and an old man of 20 stones about 12 litres—this to keep their lungs ventilated.

(5) No benefit has yet been found from the complication of a negative phase.

(6) "Patient Triggered" machines prove disappointing in use and generally recourse is needed to normal cycling. The patient must be sedated, persuaded and bullied into following the machine.

With patient triggering the following features are desirable:

(*a*) Response at less than $\frac{1}{2}$ cm. water depression.

(*b*) Immediate delivery of air.

(*c*) An open air passage so that the patient will not stifle, should triggering fail.

(*d*) An automatic cycle providing ventilation about every eight seconds should no "patient signal" be received.

(7) Oxygen enrichment up to 50 per cent. is usual and helpful in the early stages.

(8) The use of a "bath type" humidifier is universal in this country and at between 100-140° F. will deliver adequate humidity. No more is

needed than will keep secretions sufficiently moist to be removed by the sucker.

(9) Large machines of great power and frequent complexity, have their place in main centres but for the ordinary hospital it is far better to have two or more compact outfits instead.

(10) A minor comfort to the patient is a swivelling connection designed by ourselves which prevents twisting of the tube in the trachea on turning. With this, and no letting down of the tracheotomy tube cuffs, we have never had ulceration or stenosis. The tubes are changed weekly.

Transport

For transport of patients intubation is essential if bulbar symptoms have occurred and ordinary cuffed Magill tubes can safely be left in place for forty-eight hours or over. Battery operated machines are available or a 12/240 volt rotary converter can be used.

Laboratory Aids

These are of confirmatory interest but generally are too slow for the minute to minute emergencies of this technique.

Weaning

When respiratory powers return and the patient breathes unaided for longer periods certain dangers arise.

(1) Over-long effort leading to anoxæmia or exhaustion is damaging.

(2) Humidification by steam kettle or inspiring air *viâ* the humidifier is essential, or inspissated secretions may easily block the tube.

(3) Cuffs are not so effective when there is negative suction as in ordinary breathing and with persistent bulbar symptoms, great care must be taken that nothing is accidentally inhaled.

In General

(1) An experienced Nursing Staff is absolutely essential and medical advice must be constantly and instantly available.

(2) Conscious patients are always *right* when complaining of some fault in their respiration.

(3) They must be hyperventilated for comfort.

(4) Suction and percussion must always be available.

(5) A good light must be on at night and increasing vigilance by the Nursing Staff.

It is not machines but nursing which keeps these patients alive.

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BEHAVIOUR OF ABSORBABLE GELATINE SPONGE IN THE ANIMAL MIDDLE EAR

By W. DOYLE-KELLY (Dublin)

GELATINE sponge has become widely used in the following ways in otology:

1. Repair of wounds of the lateral sinus.
2. As a medium for medication in the middle ear i.e., penicillin soaked "formed drops of Wullstein", sponge soaked in corticosteroid substances or in streptokinase streptodornase solutions.
3. Supporting structures (*a*) Artificial columella.
   (*b*) Mucosal grafts within the tympanic cavity.
   (*c*) Skin grafts applied to the tympanic cavity.

These are uses for which the substance was really never invented. It was introduced primarily as a hæmostatic agent, and although the majority of the literature deals with this aspect of the material, it is worthwhile reviewing its history and the experience of others in its biological and clinical behaviour.

In the year 1911 the Boston surgeon, Harvey Cushing, described a technique for the application of a "whipped clot" to a bleeding area in a paper entitled "The control of bleeding in operations for brain tumours with description of silver clips for the occlusion of vessels inaccessible to the ligature", and suggested that "whipped clot" might be used to plaster on the bleeding surface just as he then employed solidified blood-clots and in 1916 Cushing working with fibrin, cephalin and thrombo-plastic substances described the use of "fibrin paper" as a hæmostatic in neurosurgical operations. Cushing complained that these substances had to be applied with some form of tampon, in the removal of which the clots were dragged from the hæmorraghic area.

Their studies showed that fibrin was non-irritating to tissues, less so than a muscle implant and that it was absorbed satisfactorily. In 1944 Ingraham and Bailey were responsible for an important advance in technical neurosurgery when they reported on the use of a matrix of fibrin made spongy and porous. This substance—*fibrin foam*—was soaked in thrombin and applied to the bleeding surface to obtain control of ooze from a site inappropriate to ligature, clip or electrocautery.

The result was a mesh work in which clot could form and it was found to be speedy, reliable and have little reaction to the tissues.

Oxidized cellulose was next described—surgical cotton or gauze was

converted to an organic acid by the action of nitrogen dioxide. This rendered the material soluble at an alkalinity present in tissues. Oxidized cellulose possesses a slight stypticity but as it inhibits the action of thrombin and penicillin it is classified as an absorbable pack rather than a thrombin carrier.

Starch sponge is non-irritating and absorbable but too friable.

Gelatine sponge is the substance that interests us most. It is non-specific protein devoid of antigenicity, even though Beef is the common source. It is soluble and has to be converted into an insoluble substance but an absorbable one. By whipping a lightly formalized solution a foam of uniform porosity is produced. It is cut, dried and sterilized at 160° C. Although insoluble in water it is completely digested by proteolytic enzymes. It can absorb forty-five times its own weight of blood or water, and when wet it clings to the contours of the surface to which it is applied. When wet it is non-friable, retains its tensile strength and can be safely used with both penicillin and thrombin. When buried in rat muscle fibrin and gelatin sponge disappear in fifty and thirty days respectively, the reaction being equal.

Experimental work with gelatine sponge now falls into two categories namely:

(1) The earlier work of Americans whose attention was focused mainly on the hæmostatic capabilities of the material in conjunction with thrombin and

(2) More recent work on absorbability of gelatine sponge *per se* done in England.

With reference to ear surgery our attention is more focused on the behaviour pattern of the absorbtion of the sponge as used for support of material that would otherwise have to bridge space. However, a short review of the American work will not be out of place.

Light and Prentice writing in the *Archives of Surgery*, September, 1945, report on the use of sponge in a series of twenty-four neurological operations. They compared the patch technique with the repair of a motor tube. The gelatine sponge is the patch, the thrombin the vulcanizing agent. The patch is rendered impervious after it becomes soaked in blood. A thin patch is adequate for minor ooze, but a thicker one is required to stem a more vigorous flow. The material is immersed in thrombin solution 100 units per c.c., kneaded, air expressed and resoaked prior to application.

Working on the animal they made various types of implant; on the surface of the intact brain, on the bleeding brain, deep in brain tissue and on torn venous sinuses. They observed that the sponge implant provoked a predictable mild cellular reaction and was absorbed in three to five weeks without leaving residual reaction. Cellular reaction commenced at *one week*, was maximal at *two weeks*, with lymphocytic infiltration and giant cell formation on the dura. After *three weeks* breaking up of

the material commenced. After *four weeks* reaction was negligible and the ultimate glial reaction was no worse than expected by injury alone. Furthermore, repair of the venous wall left the lumen intact. In no case was thrombus formation seen.

Pilcher and Meacham (1945) of Nashville, Tennessee, in an article entitled "Absorbable Gelatine Sponge and Thrombin for Hæmostasis in Neurosurgery", exposed the superior sagittal sinus in dogs, removed strips of venous wall 1 mm. wide, applied the patch and examined their material at intervals, varying from two days to six weeks post-operatively. They described: early infiltration with polymorphs into the sponge spaces:

1 week—fibroblasts and round cells replace early inflammatory change.

2 weeks—changes more pronounced.

Later—absorption of material with the formation of new blood vessels.

4 weeks—material almost disappeared. Young vessels still present. Fibrous tissue dense.

6 weeks—site of implant has healed with plaques of fairly dense elastic tissue remaining.

The sinus remained patent in every case.

They furthermore analysed the experience of their colleagues' cases. Out of 291 neurosurgical operations, including 41 of their own, the result where the technique was used was declared satisfactory in all but eight, and of these eight, 5 poor results were not attributable to the sponge. Among a list of uses given by these authors, those that would seem to appertain to ear, nose and throat surgery, are uses involving the control of ooze in tumour bed, control of epidural venous bleed from under bone edge and in the preparation of a dry bed for suture of peripheral nerves. Writing in the following issue of the same Journal, Correll and Prentice (1945) report on the findings in a series of implants of thrombin soaked gelatine sponge into the leg muscle of rats and they stated that absorption was complete by the 30th day, without the tendency to encapsulate that was seen with occasional cases of fibrin sponge implants.

They maintained that the cellular infiltration provoked was considered to be no greater than occurring with the resorption of ordinary blood clot. The cellular reactions were found to be maximal in 10-25 days and then recede.

The *Lancet* of September 9th, 1951, issued an article on absorbable gelatine sponge in experimental surgery written by George Blaine, and this worker used the material without a hæmostatic agent. He implanted the material into three sites:

The abdominal cavity, where it was readily enveloped by omentum.

The muscle of the anterior triangle of the neck.

Into the incised liver wound.

The animals were examined and reported on as follows:

	Macroscopic	Microscopic
5th day	Readily found by naked eye. Firmly adherent.	Acidiphilic: amorphous: surrounded by polymorphs. (Early inflammation.)
11th day	Easily found, smaller adherent fragmented.	Subsiding inflammation fibroblasts and round cells present, with giant cells advanced stage of digestion and basophilic.
16th day	Very few fragments	New blood vessels, fibrous tissue, giant cells, some fragments.
23rd day	Still palpable.	Fibrosis—but no more than could be expected in an unimplanted wound.
28th day	Not palpable	Fragments difficult to identify; dense linear fibrous tissue, no encapsulation.
42nd day	—	Fibrosis subsiding; no identifiable fragments of sponge were seen.

Blaine pointed out that hæmostasis was satisfactory without thrombin, that penicillin was not inhibited, that if too much sponge was used absorption does not take place satisfactorily because access to phagocytosis in the centre of implant is prevented. Absorption or organization took place in 4-6 weeks.

Before proceeding to a description of the present work undertaken on the rabbit middle ear two references to continental literature may be worthwhile.

(1) Viikari and Larmi (1951) implanted prepared Gelatine Sponge into the omentum, liver and muscle tissue of rabbits and stated that a small remnant was observed in muscle tissue in a period up to forty days, but complete absorption had taken place in seventy days. In the liver no trace of implanted material could be found after thirty days. Furthermore they observed that the hæmostatic effect of the sponge did not differ whether it was used in a dry state or whether it was wetted with saline and then squeezed dry.

(2) Sigurd Rauch (1958) has used gelatine Sponge as a substitute to replace tissue removed at operation and reported on its use as an implant after radical parotidectomy. He found that in human beings, formalized gelatine sponge was tolerated much better than in rodents. In the human resorption in connective tissue was completed in the course of a fortnight approximately, while in rodents it took four to six weeks.

The present work was undertaken in the Department of Physiology, Trinity College, Dublin, and the animal used was the rabbit. Anæsthesia was induced and maintained by intravenous injections of Nembutal solution, in accordance with a schedule worked out cc./Kg. body weight. Under aseptic technique, post-aural incision through the shaved and

prepared skin of the animal was made, bone over mastoid exposed and entrance made to the middle-ear postero-superior to the tympanic ring in much the same manner as one would make an attico-antrotomy. Using a dental drill of small dimensions with the help of suction and irrigation under five times magnification, the middle ear was then examined through the drill hole. If the incus presented much in the way of obstruction it was removed through the operative defect. A small window was cut in the cartilaginous meatus near the drumhead for further inspection. Sterispon was packed in, in strips, so much as could be got in, the middle ear was then flooded with penicillin solution 10,000 u/cc. The cortical defect and muscles packed and two layer closure made. Animals were killed for examination at the following intervals following operation:

3 days—

1, 2, 3, 5, 6, 7, 9 weeks.

The specimen was perfused and prior to having its temporal bone dissected out, was fixed in formaldehyde solution. Blocks were embedded in paraffin in some cases, and in low viscosity nitro-cellulose in others. Sections cut were stained in hæmatoxalin and eosin.

Fig. 1. Shows normal mucosa of a control animal. Mucosa is a single cuboidal epithelial layer, but in some places it may be low columnar and two or three layers thick.

Fig. 2. Is that of a three-day implant and demonstrates the gelatine sponge in the middle ear as a basophilic mesh, in part of which strands are visible. The meshes are crowded with cells. The material is in contact with the muco-periosteum, although no local abrasion had been made.

Fig. 3. The majority of the cells are polymorphs, which infiltrate the lining and invade the implant. Odd macrophages are seen on detailed examination.

Fig. 4. In some parts where the material is sparse the cellular reaction is scanty.

Fig. 5. The lining mucosa has become markedly thickened, the cells forming many layers. As well, there is increased vascularity in the membrane. This thickening is shown in higher power in Fig. 6.

Fig. 7. Demonstrates the malleus with similar surrounding reaction.

In the one-week implant the mucosa is again thickened, as shown in Fig. 8.

Fig. 9. Cellular response is very largely polymorphonuclear. Fibrosis is commencing.

After *two weeks* many polymorphs are still present, but multinucleated giant cells are seen and these are very numerous in places. Fibroblasts are also evident. The epithelium can be seen to reach out to the implant, as seen in Fig. 10. Cellular reaction is particularly marked at the outer side of the sponge.

FIG. 1.

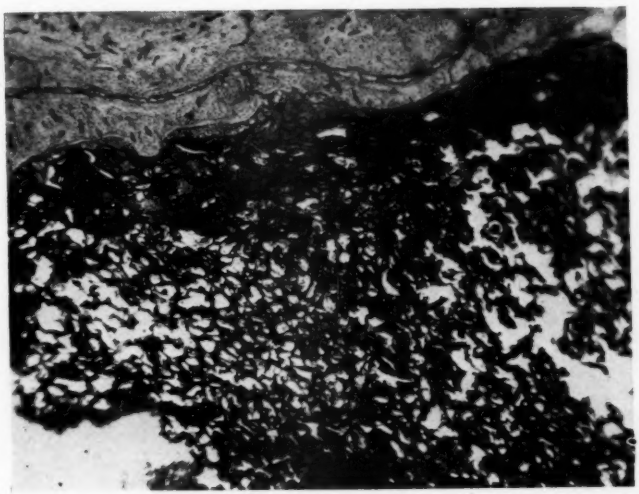

FIG. 2.

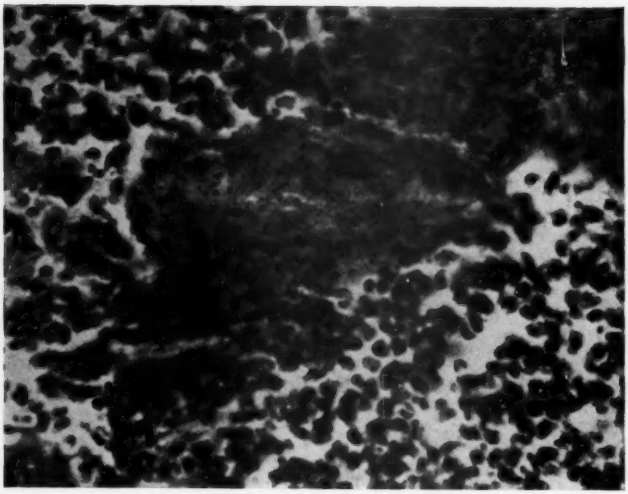

FIG. 3.

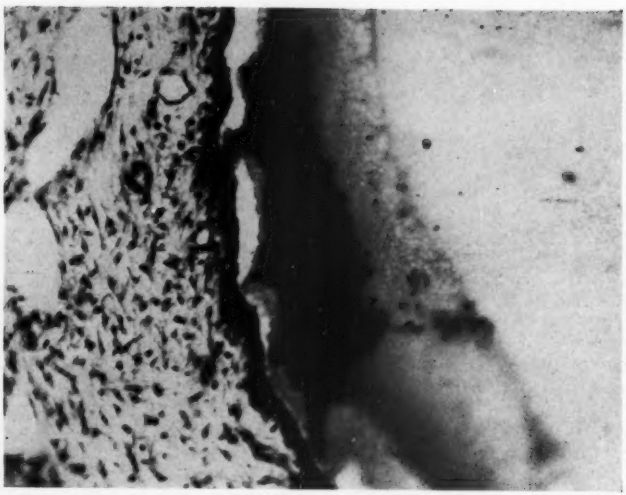

FIG. 4.

FIG. 5.

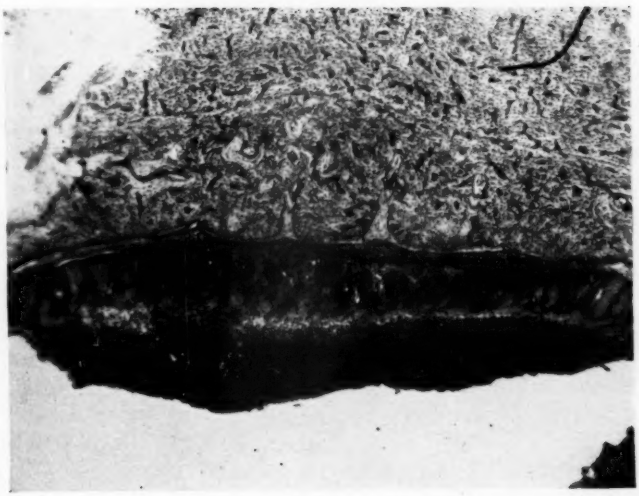

FIG. 6.

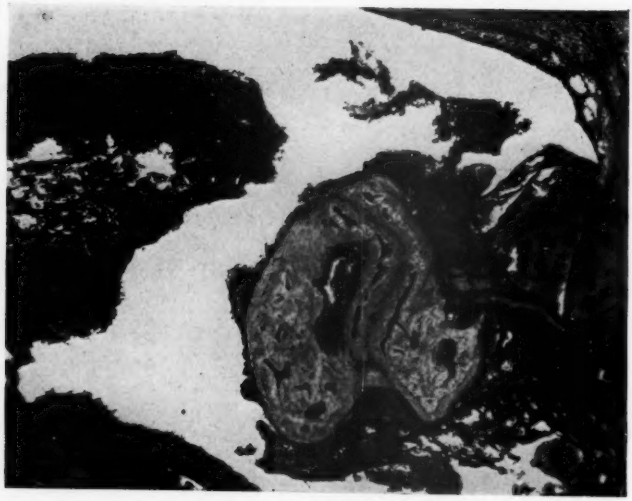

FIG. 7.

FIG. 8.

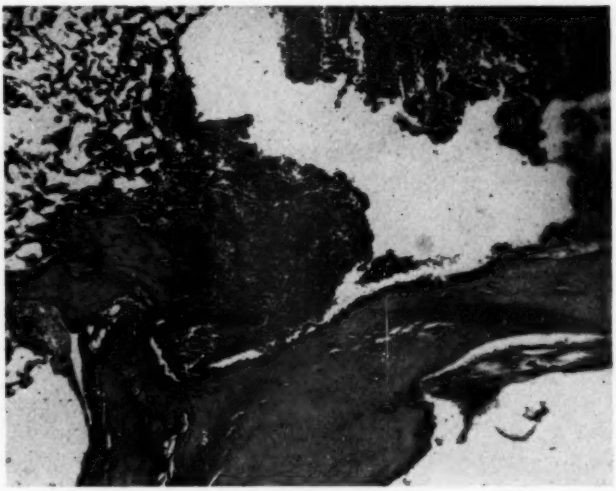

FIG. 9.

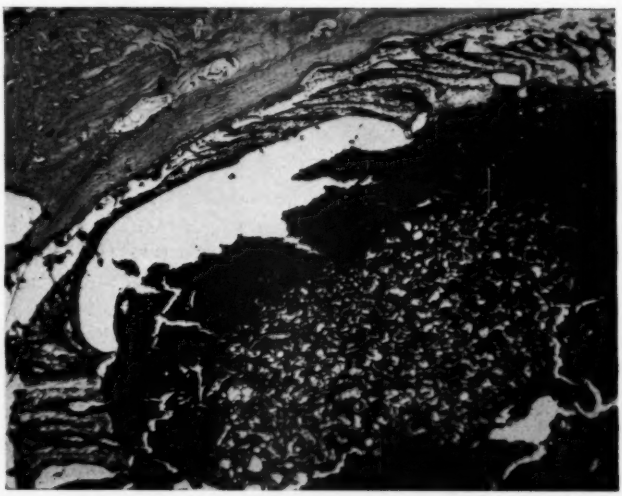

FIG. 10.

After *three* weeks polymorphs are fewer, but large round cells, histiocytic phagocytes are seen. Fig. 11 shows well-marked fibres of gelatine sponge with cellular clumps, enmeshed. Fibroblasts are present and proliferating in the cavity. Epithelium is thickened in places and there is glandular hypertrophy in the areas where glands are present. Œdematous submucosa and epithelium seem to be probing their way into the implant carrying in fibroblasts and vessels. This is well seen in Figs. 12 and 13.

The five to seven week implants characteristically show proliferating fibrous tissue. Fibroblasts are active and collagenous tissue evident. The tissue within the middle-ear cavity contains very numerous, large, dilated new capillary blood vessels. Odd pieces of sponge were visible within the mass of the proliferating connective tissue of the five-week impant but not within the seven-week one. Figs. 14 and 15 show the five-week implant in low and high power respectively.

Curiously enough, in the second series, a six-week implant shows no trace of intra-tympanic gelatine sponge. There were no round cells visible—no scar tissue. The lining was thickened in places and a fibrinous deposit was present against the mucosa in certain sites. The explanation of this is possibly due to overfilling of the middle-ear space with sponge in the five- and seven-week specimens.

Finally a *nine-week* implant was made and the middle ear filled well with sponge. On examination the cavity is devoid of material, cells and fibrosis. The mucosa has thinned out to almost normal proportions and no glandular hypertrophy was evident—Fig. 16.

Conclusions

Removal of absorbable gelatine sponge takes place from the middle ear of the experimental animal (rabbit) by a process of phagocytosis followed by organization and finally digestion and complete removal. The changes seen in the middle ear are similar to the changes seen in other tissues by previous workers.

Removal through the eustachian tube did not take place.

It was found that absorption takes place earlier in cases which are lightly packed, i.e., about six weeks, than those cases in which packing is more firmly placed. In either case no intra-tympanic fibrosis or obliteration took place, nor was there any residual damage to the lining membrane.

From the amount of connective tissue hyperplasia it appears that the addition of proteolytic enzyme or cortico-steroid solutions may be advisable as a solvent for the sponge, or there may be a case for the administration of cortico-steroid by mouth some weeks after operation in the human.

FIG. 11.

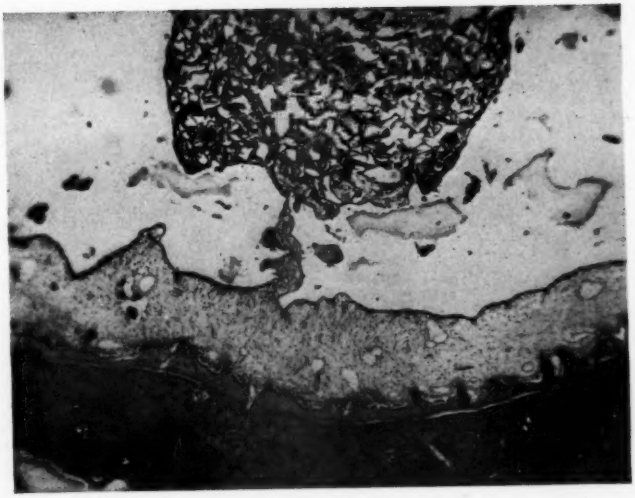

FIG. 12.

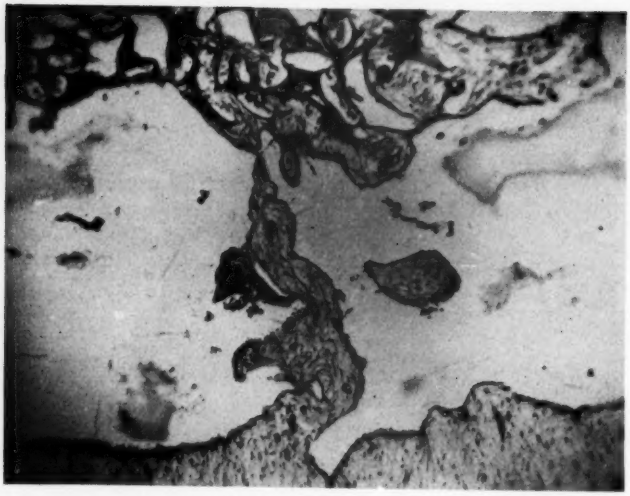

FIG. 13.

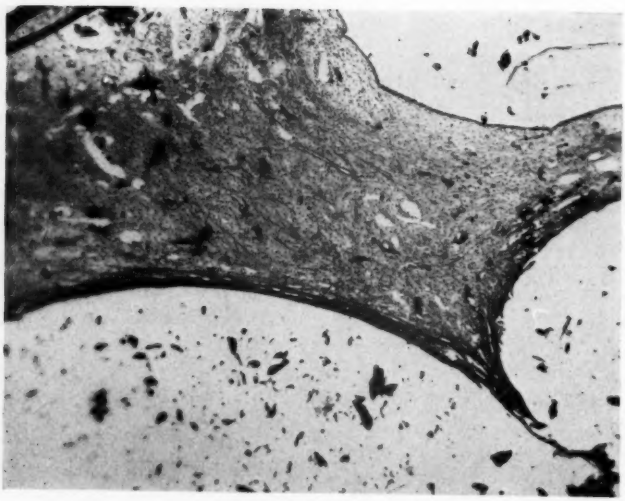

FIG. 14.

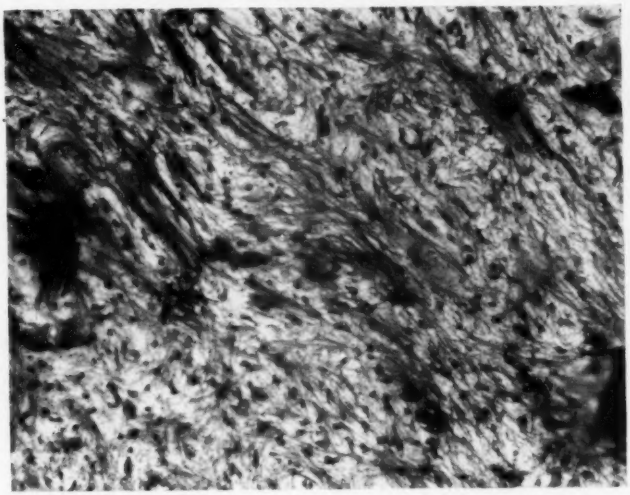

FIG. 15.

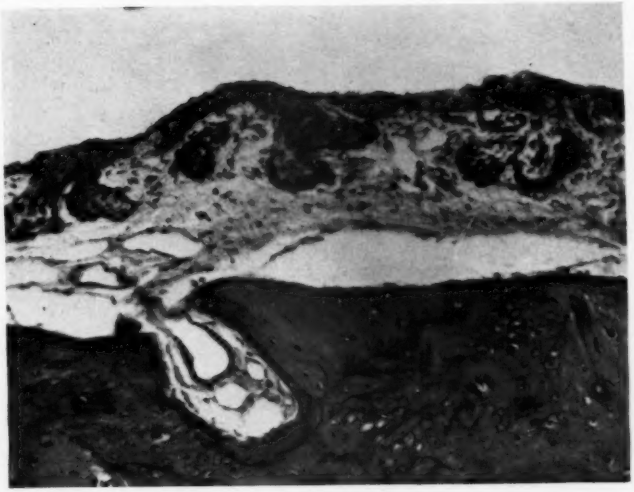

FIG. 16.

Acknowledgments

I wish to thank Professor D. S. Torrens for the facilities he offered me in carrying out the above work, and in interpreting cellular reactions. I would also like to thank Mr. Oliver Murphy, M.R.C.V.S., Mr. L. Wilmot all of the Physiology Department, Trinity College Dublin. Mr. F. A. Murray for the photomicrographs and Doctor G. R. Boyes of Messrs. Allen and Hanbury Limited, for supplies of the Gelatine Sponge.

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THE SIGNIFICANCE OF MYCOTIC INFECTION IN THE ÆTIOLOGY OF OTITIS EXTERNA*

By A. E. W. GREGSON and C. J. La TOUCHE (Leeds)

THIS paper is a preliminary report of the results of an investigation which Dr. La Touche and I have carried out over the last four years.

Like most E.N.T. departments, we have in Leeds a number of cases of chronic otitis externa. Either they show no response to treatment, or, if they do respond, they very soon relapse. Many explanations have been offered for this problem, but it seemed to us that the possibility of fungus infection has been neglected.

With this in mind it was agreed that Dr. La Touche should investigate examples of this type of case, and where an actively growing fungus was found treatment with a fungicidal agent would be given.

As can be seen from these figures the practice has grown.

TABLE I.

NUMBER OF PATIENTS EXAMINED FOR OTOMYCOSIS

Year	Total examined	Total positive
1956	7	5
1957	19	8
1958	20	11
1959	134	59

The figures for 1959 do contain fourteen examples of fungus infection in Radical Mastoid or Fenestration cavities, but it was thought that the problem was sufficiently similar to justify their inclusion.

These figures may cause some surprise, although mycotic infections elsewhere have become more common with the advent of antibiotics. And it must be emphasized that Otomycosis has only been diagnosed when the fungus has been seen to be growing actively in a scraping of debris taken from the auditory meatus—at least the mycelial form, or, better still, the spore-bearing conidiophores. Merely to take a swab from the meatus and subject it to culture is not enough. Fungus spores abound in the atmosphere and may readily contaminate the plate or swab. As a control we

* Paper read at the Otological Section, Royal Society of Medicine, London, February 5th, 1960.

have examined scrapings taken from the clinically normal and symptom-free meatus of sixteen volunteers from the hospital staff. None gave evidence of a fungus, but subsequent cultures from swabs gave positive results in two cases—thus illustrating the unreliability of swab technique.

The fungi most commonly found have been the Aspergillus and Candida species.

TABLE II.

TOTAL NUMBERS OF FUNGI ISOLATED

1956	*A. flavus*	..	..	2	1959	*A. fumigatus*	..	..	13
	A. niger	..	..	2		*A. niger*	..	..	12
	Candida Sp.	..	..	1		*A. nidulans*	..	..	1
	Yeasts	..	..	1		*A. terreus*	..	..	3
1957	*A. fumigatus*	..	..	1		*A. herbariorum*	..	..	1
	A. niger	..	..	3		*C. albicans*	..	..	12
	C. albicans	..	..	2		*C. parakrusei*	..	..	1
	C. krusei	..	..	1		*C. guillermandii*	..		1
	C. parakrusei	..	..	1		*A. niger and C. albicans*			1
1958	*A. fumigatus*	..	..	1		*A. candidus* Sp.	..		1
	A. niger	..	..	3		Yeast Sp.	..	..	10
	A. nidulans	..	..	1		*S. brevicaulis*	..	..	1
	Scopulariopsis Sp.		..	1		Actinomycetes	..	..	2
	C. albicans	..	..	4					
	Candida Sp.	..	..	1					

Although the Aspergillus has been recognized as the cause of Otomycosis there has been little mention of Candida previously.

Clinical Picture. At first one had no idea of the clinical picture. Descriptions in books of reference were brief and uninformative. Patterns are, however, becoming apparent, but I must emphasize that, even now, they are nothing more than impressions, and clinical diagnosis is unreliable.

One symptom has occurred constantly. This is a sense of irritation in the meatus which may be well-nigh intolerable. Pain is unusual without an associated pyogenic infection. Some infections in Radical Mastoid cavities do seem to cause severe pain. (We are indebted to Mr. Capps for drawing our attention to this.) Discharge is scanty and not fetid—usually quite odourless. Deafness in the non-operated meatus is unusual.

The meatus is rarely swollen unless self-inflicted trauma has led to a secondary pyogenic invasion. In the acute stage it is filled with a mass of grey, caseous debris quite unlike the "wet blotting-paper" of classic descriptions. Removal of this debris shows a meatal lining of a distinctive magenta colour, covered with small masses of glistening white debris. The tympanic membrane, while discoloured, is of normal contour.

Less acute cases may present two patterns, depending on the type of infection. Where Aspergillus is present the meatus is notably dry and the magenta colour is less evident. Clusters of conidiophores may be seen, resembling miniature pin-heads. They may be white, yellow, brown, black,

or green, depending on the age and species of Aspergillus. Mr. R. R. Simpson has said that he found this picture frequently among native troops in India without any symptoms of ear disorder, but I have only seen this occur once in a European.

In Candida infections the picture more nearly resembles the acute case after cleansing. The masses of fungus can be seen in these cases and the tympanic membrane is covered with them.

It is difficult to recognize these patterns with the naked eye. At least the magnification of the electric auriscope is required. Moreover the picture can be modified out of all recognition by previous treatment, particularly if greasy preparations have been used.

From the diagnostic aspect there are three features which should arouse the suspicion of a mycotic infection.

(1) Relapse after, or resistance to, standard treatment.

(2) The symptom of irritation.

(3) A history of local antibiotic therapy.

Thereafter one is dependent upon the recognition of actively growing fungus by the methods outlined.

Treatment

These cases have been treated with Nystatin (Squibb and Co.) in order to assess the value of this new fungicide. Of the eighty-three proven cases, two refused treatment, seven cannot be traced, and seven suffered relapses which responded to further periods of therapy with the same drug. The remainder responded to the initial period of therapy.

Treatment is given three times weekly for a period of at least three weeks. At each attendance the meatus is meticulously cleansed by dry mopping and its epithelium is lightly frosted with boric powder containing Nystatin. A concentration of 100,000 units of Nystatin per gram was used at first. Recently we have found 1,000 units per gram effective.

Cure is accepted when the meatal epithelium is free of debris, of normal colour, and is showing a little early secretion of normal wax.

Apart from this apparently very satisfactory curative quality, Nystatin has two other advantages. Its application is painless, and it does not obscure a view of the tympanic membrane and meatus. It must be admitted, however, that ten cases in this series did develop a furunculosis while under treatment. The explanation of this is in doubt. Cross-infection by the "hospital staphylococcus" is a possibility, but there is some evidence that penicillin-resistant staphylococci and fungi are the most usual contaminants after periods of antibiotic therapy and many of these patients had received such therapy previously. Since many of the fungi which have been isolated are known to produce efficient antibiotics it is thought that the development of a furunculosis is a fair indication that

fungi have been eliminated. So far it has responded satisfactorily to simple measures such as local 10 per cent. Ichthyol in Glycerine.

There are, of course, many other possible methods of treating Otomycosis, but most involve some undesirable reaction. Tincture of Iodine, possibly the oldest, causes intense pain when it is applied, and I suspect that many patients defaulted from treatment, thinking it worse than the disease. Whalen admits that his Thymol in Metacresyl Acetate is open to the same objection.

The aniline dyes are certainly efficient fungicides, but their colour obscures the clinical picture, and it must be well-nigh impossible to recognize a cure in the deep purple gloom of a meatus treated with Gentian Violet.

It has been suggested in the past that otomycosis responds rapidly to the use of Industrial Spirit topically. This is open to grave doubt, as experiments in culture media show. The effect of Gentian Violet and of Nystatin is apparent from the wide ring of inhibition in each culture. The B.N.F. standard 50 per cent. Industrial Spirit appears to have no fungicidal effect at all.

Conclusion

One would not pretend that fungus infection is the complete explanation of chronic otitis externa. Quite obviously it cannot be when over 50 per cent of the cases investigated have not shown its presence. Nevertheless a search for mycotic organisms and their elimination by specific therapy does seem well worth-while. These patients very easily become "constant attenders" and it has been a relief, both to them and ourselves when their names disappear from the Outpatient lists.

Acknowledgements

We are indebted to Mr. T. McM. Boyle, Mr. O. C. Lord, Mr. C. P. Mills, and the late Mr. G. S. Seed for permission to make this investigation on patients under their care. We would like to thank Mr. A. L. Pegg of the University of Leeds Medical Photography Department for his help and advice, and Mr. R. A. Forster, Technician to the Mycology Section of the Department of Dermatology, United Leeds Hospitals for the preparation of cultures and specimens.

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CLINICAL RECORDS

DEAFNESS IN TOXOPLASMOSIS

By MOSHE FEINMESSER and JACOB LANDAU (Jerusalem)*

TOXOPLASMOSIS, an infection due to Toxoplasma gondii, may appear in either congenital or acquired forms.

The clinical manifestations of congenital toxoplasmosis were described by Woods (1956) as: "either macular or extra-macular focal choroidal exudates, encephalitis, often internal hydrocephalus, mental retardation, various other neurological signs, and often areas of cerebral calcification".

Most cases of the acquired form of toxoplasmosis are asymptomatic. Focal choroiditis occurs only occasionally, and systemic symptoms are rare. A positive serological reaction (Sabin-Feldman dye test) is usually the only evidence of the infection.

There are few published reports of ear involvement in this disease (Callahan *et al.*, 1946, Campbell and Clifton, 1950, Frenkel and Friedlander, 1952, Dietzel, 1958, Kelemen, 1958), and it, therefore, seems worthwhile to report the following case in which congenital toxoplasmosis was associated with deafness.

Case Report

A.R., a 14 year old Yemenite boy suffering from severe deafness, had been in a school for the deaf from the age of 7 years. He was the oldest child; the four other children were of normal development and possessed good hearing. His general physical development seemed normal, but when he had not even "burbled" by the age of 2 years his mother suspected deafness. In early infancy he had suffered from a transient discharge from the ears. Two years after admission to the school for the deaf, defective vision was also suspected, and the patient was examined by an ophthalmologist.

The general physical examination revealed no abnormalities, except for those of hearing and vision. The results of the examinations of the urine and blood chemistry were within normal limits. Blood counts on two occasions showed an eosinophilia of 5 per cent. and 9 per cent. The Wassermann and Kahn tests were negative, as was the Mantoux test.

X-ray examination of the skull revealed no calcifications or other abnormalities except for a slightly sclerotic right mastoid process. The E.E.G. was normal.

The Sabin-Feldman dye test was positive to a titre of 1:64.

Ophthalmologic findings: There was a large, round, chorioretinal lesion in the macular region of the fundus of the right eye. The lesion had sharp margins,

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was mostly pigmented, and contained atrophic areas (Fig. 1). The left fundus was normal. Vision was:

O.D.—$0 \cdot 5^{\wedge}_{\vee}$ cyl. + 1·0 ax. 90° 5/20.
O.S. cyl. + 1·0 ax. 90° 5/6.

The findings were similar to those of the first ophthalmologic examination when the patient was 9 years old. The diagnosis made was non-active, right

FIG. 1.
Right eye: Large chorioretinitic lesion in the macular region, with scarring.

chorioretinitis centralis with scarring. The lesion described is characteristic of toxoplasmosis.

Otologic findings: Both ear drums were normal. An audiometric test revealed bilateral subtotal nerve deafness (Fig. 2). There was no vestibular reaction even to strong stimuli during caloric and rotation tests.

All the other members of the patient's family were examined. In none of them was any abnormality detected on general, ophthalmologic and otologic examinations. The Sabin-Feldman dye test was positive to a titre of 1:64 in the mother, negative in the father and siblings. On the basis of the serological finding the mother was diagnosed as suffering from asymptomatic acquired toxoplasmosis.

Discussion

Association of nerve deafness with toxoplasmosis of the eye was reported by Campbell and Clifton (1950) in 3 members of the same family. The Sabin-Feldman dye test was positive to a titre of 1:128 in one of them, and to a titre of 1:32 in the other two. Dietzel (1958) found slight hearing disturbances in 5 out of 10 children affected by toxoplasmosis. The diagnosis was proved by the

presence of the parasites in the spinal fluid. The author concluded that toxoplasmosis could be a cause of hearing defects of retrolabyrinthine origin. The histopathologic changes in the ears of a 25-days old infant, and of a fetus delivered following perforation because of hydrocephalus, were described by Kelemen (1958). In the first case toxoplasma parasites were demonstrated, whilst in the second the pathological findings suggested toxoplasmosis. He emphasized that infantile toxoplasmosis must be considered a hitherto unrecognized cause of congenital hearing defects. Callahan, Russell and Smith (1946) reported a case of a 6-weeks old boy on whom a bilateral mastoidectomy

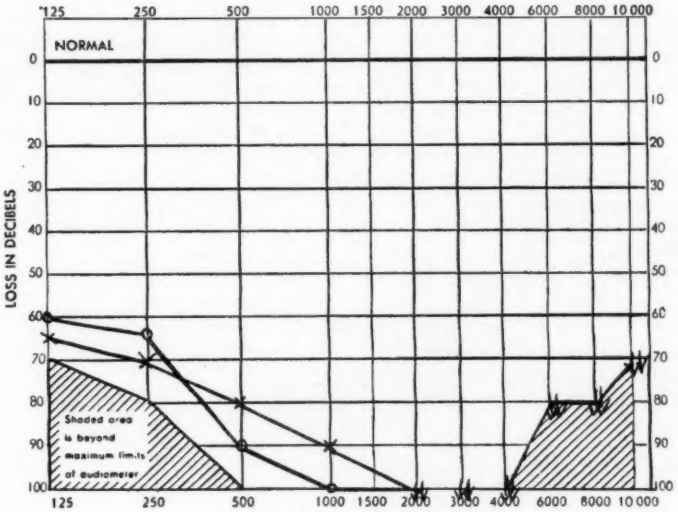

FIG. 2.
Audiogram, indicating subtotal bilateral deafness.

was performed. Death occurred 20 days later. Toxoplasma parasites were identified in histological sections of the left middle ear. Wilder (1952) found the parasites in the sections of 53 human eyes.

A positive Sabin-Feldman dye test to a titre of 1:64 is considered of diagnostic value (Beverly and Beattie, 1954, Woods *et al.*, 1954). In the case of the boy described above, the lack of evidence of any other granulomatous disease to which the choroidal scars might have been attributed also favours the diagnosis of congenital toxoplasma choroiditis. The discharge from the ears in infancy was probably due to a common infection leading to otitis media, but toxoplasmosis cannot be excluded as the cause. It does not seem likely, however, that the nerve deafness was a result of the middle-ear infection. The evidence suggests that both the fundus of the eye and the hearing apparatus were simultaneously injured by the toxoplasma infection.

Summary

A case of congenital toxoplasmosis is described in which there was toxoplasma choroiditis in one eye, and bilateral subtotal nerve deafness.

The possibility that the deafness was due to the toxoplasma infection is discussed.

Acquired asymptomatic toxoplasmosis was diagnosed in the patient's mother on the basis of a positive Sabin-Feldman dye test.

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TRACHEAL DIVERTICULA

By C. H. JONES (Liverpool)

DIVERTICULA of the trachea have been recorded only rarely. While they may occasionally assume clinical significance, for example, by pressure on the recurrent laryngeal nerve, or by becoming infected, the majority have been discovered post-mortem, although some have recently been visualized radiographically during life. The lesions in the case to be described were incidental findings at necropsy in a man of 55 who died a few days after an extensive resection for a carcinoma of the cardia.

Macroscopic Appearances (Figs. 1 and 2)

The larynx was normal. An ovoid cyst measuring 3·5 × 3·5 × 3 cm. arose by a pedicle 1 cm. in diameter from the right postero-lateral border of the

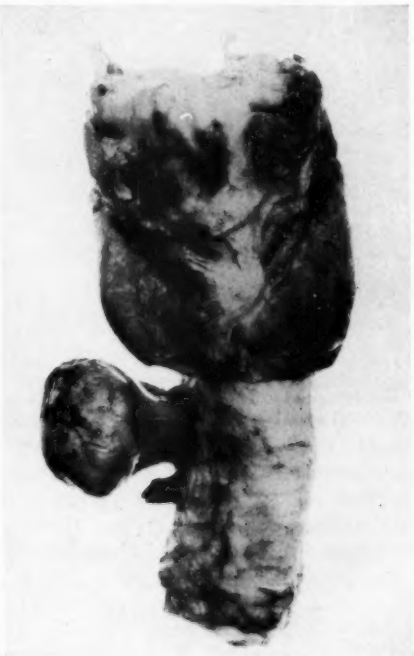

FIG. 1.

Photograph of the gross specimen, after fixation, viewed from the front.

trachea, its upper border 4·5 cm. below the vocal cords and 1·5 cm. below the lower pole of the thyroid gland. Its wall was firm and easily separable from the surrounding tissues, and the cyst communicated with the trachea by an orifice, through which a fine probe could just be passed, situated at the junction of the cartilaginous rings and the trachealis muscle, and between

FIG. 2.
Posterior aspect of the same.

two adjacent cartilages. On gross section the cyst was empty and unilocular, and its wall varied in thickness up to 6 mm.

Immediately below this there was a second cyst, measuring 1 × 0·8 × 0·5 cm., which communicated with the trachea by a small opening in line with the previous one, and in the inter-cartilage space next below it.

Apart from these lesions the trachea and bronchi appeared macroscopically normal.

Microscopic appearances

The larger cyst is almost completely lined by a stratified ciliated columnar epithelium which appears healthy (see Fig. 3). Immediately external to this is a layer of loose connective tissue infiltrated by lymphocytes, plasma cells

and a very occasional polymorph leucocyte. The remainder of the wall is composed of laminated collagen fibres in which a few capillaries and arterioles are present. No cartilage or smooth muscle can be seen. Throughout the thickness of the wall there are many irregular duct-like structures with an epithelial lining similar to that of the main cavity. A few of these spaces contain inspissated mucoid material but the majority are empty, and all are surrounded by

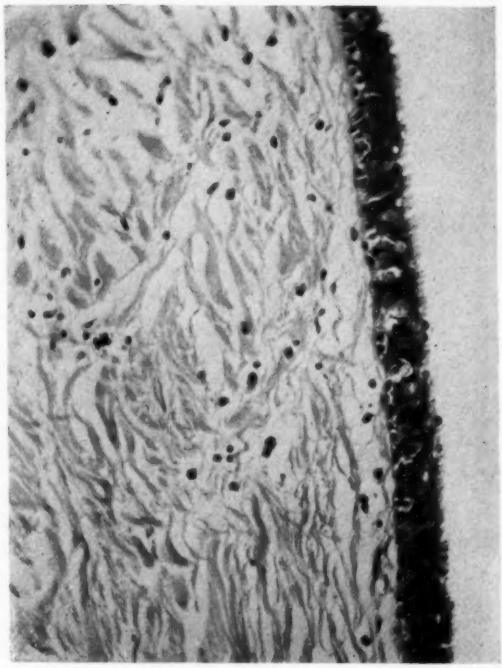

FIG. 3.

Photomicrograph of the wall of the larger diverticulum showing the stratified ciliated columnar epithelium, and part of the fibrous layer. There is a mild inflammatory infiltration beneath the mucous membrane (Hæmatoxylin and Eosin. ×220).

an infiltration of chronic inflammatory cells. No mucous glands are present in the wall.

In the smaller cyst the epithelium is similar to that in the larger, but the fibrous wall is thinner, more vascular and less collagenous. There is a light round-cell infiltration. No cartilage or muscle is seen but several apparently normal mucous glands are present (see Fig. 4).

A tranverse section through the openings into the trachea shows them to be lined by healthy ciliated columnar epithelium. In their immediate vicinity the mucous glands of the tracheal wall have distended ducts filled with inspissated material. Sections from the wall of the trachea at other sites show a

mild sub-mucosal round-cell infiltration and moderate hyperplasia of mucous glands which are otherwise normal.

Discussion

Very rarely diverticula arise from the anterior wall of the trachea in the median line close to the thyroid gland. These so-called tracheocœles may represent congenital herniae of the mucous membrane, or may result from

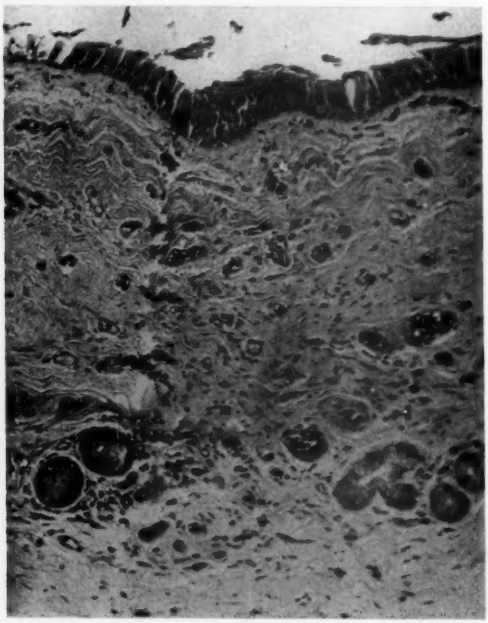

FIG. 4.

A section from the smaller diverticulum showing a similar structure to the previous figure but with mucous glands in the wall (Hæmatoxylin and Eosin. × 120).

chronic inflammation of the trachea with, or without, there being a congenital weakness of the wall. Pouching of the whole or the greater part of the posterior wall in association with emphysema has also been described. Neither of these varieties will be discussed further.

The majority of tracheal diverticula arise from the right side. It is clear from the literature that, ætiologically, two different lesions occur, the one acquired and the other congenital. The number of cases recorded, of both varieties, is very small and a summary of them will be found in the paper by Goldman and Wilson (1957).

The lesions in the present case are examples of the acquired variety. These were probably first described by Rokitansky (1838, quoted by Schwalbe 1912),

who regarded them as retention cysts of mucous glands. The only series of cases published is that of MacKinnon (1953), who described ten. In three of these there were two cysts, bearing the same relationship to each other as those in the present case, while in the remainder the lesions were single. The cysts varied from 0·5 to 3 cm. in diameter, and all were attached to the right postero-lateral border of the trachea, with which they communicated by a fine opening situated at the junction of the cartilaginous rings and the membranous part. The upper border of the cysts lay just below the lower pole of the thyroid gland in all but one instance where the lower border was 2 cm. above the tracheal bifurcation. In most cases a vertical series of small openings, which histologically were the dilated ducts of mucous glands, was present on the right side of the trachea above and below the orifices of the cysts. The cysts were either unilocular or multilocular, and histologically all were similar in having a stratified ciliated columnar lining, and a wall of fibrous tissue from which cartilage and muscle were absent. A chronic inflammatory infiltration was present in all, and mucous glands were found in the wall in two cases. In this series seven were male, mostly in the older age groups, and all but one had a history of a chronic cough. The exception was a man of 32 with rheumatic valvular disease, who differed also from the others in that the cyst was close to the tracheal bifurcation. MacKinnon considered the diverticula in his series to be acquired lesions, and agreed with Rokitansky's view that they arose by cystic dilatation of the ducts of mucous glands. He thought the earliest stage to consist in dilatation of the ducts with hyperplasia of the glands, which was followed by further cystic distension of the ducts with associated atrophy of the glands, there finally remaining a simple epithelium-lined fibrous-walled cyst.

An alternative explanation for their origin was put forward by Przewoski (1898 quoted by Mathey and Lemoine 1954) who, while accepting their acquired nature, suggested that they were pulsion diverticula resulting from degeneration of the elastic and muscle fibres of the posterior tracheal wall.

MacKinnon's principal reason for believing that the cysts were derived from mucous glands was that he found dilated gland ducts in other parts of the trachea, and assumed that these were the earlier stages in the formation of the cystic lesions. This, however, is purely speculative. The usual cause of distension of a duct system is obstruction of its outlet, but with continuing secretion by its glandular cells. The cysts in question do not give the impression histologically that this is their mode of formation, since there is no obstruction of their outlet or evidence or retained secretion, and since the mucosa is healthy, unlike the flattened, atrophic lining usually found in cysts formed in this manner. Moreover, hyperplasia of tracheal glands with dilatation of their ducts is the rule in chronic bronchitis. The incidence of acquired diverticula is difficult to assess. Very few cases have been reported, but on the other hand eight of MacKinnon's cases when the lesions were being specifically sought, were found in 867 consecutive necropsies. Nevertheless, in view of the high incidence of chronic bronchitis it seems probable that some other mechanism must be operative in the production of the diverticula. A herniation of the tracheal mucosa through a congenital or acquired defect in the tracheal wall seems a more likely explanation of the histological appearances of these lesions. Miller

(1913) described the trachealis muscle as being composed of transverse bands between which there are relatively amuscular areas which might serve as the site of election for diverticula. The spasmodically high intra-tracheal pressure of a chronic cough might then serve to expose a congenital weakness, or on the other hand the changes of chronic bronchitis might lead to degeneration of the elastic tissue of the tracheal wall. The restriction of acquired diverticula to the right side of the trachea is probably explained by the fact that the œsophagus deviates slightly to the left and thus leaves the right edge of the membranous trachea relatively unsupported. Whatever their mode of production, their acquired nature is suggested by their being found for the most part in elderly men with a chronic cough. The patient in the present case had had a cough for 30 years.

To be distinguished from the lesions already described, on grounds of both ætiology and structure, are those whose congenital nature is undoubted. These were first described by Chiari (1889) in a series of five cases. They too are found on the right side of the trachea and also open into the lumen at the junction of cartilaginous and membranous parts, but often nearer the bifurcation. They are found at all ages and are more common in males. Their principal histological difference from the acquired diverticula is the presence in their wall of cartilage and muscle. Their pathogenesis is in an aberration of that process of tracheal budding in the early embryo which gives rise to the right eparterial bronchus. In its more developed forms the abnormal bud becomes a tracheal bronchus or supplies an accessory lung, but if the process is abortive a diverticulum of the trachea results. If the abnormal bud ends blindly and loses its connection with the trachea, there may result a mediastinal bronchial cyst, also found on the right side, as described by Laippley (1945). Developmental abnormalities of the bones of the thorax or of the great vessels may be associated with congenital tracheal diverticula.

Summary

The gross and microscopic appearances of two tracheal diverticula, found incidentially at necropsy in a man of 55, are described. A distinction is drawn between acquired and congenital diverticula, and the pathogenesis of each discussed. The lesions in the present case are considered to be of the acquired type.

Acknowledgements

I wish to thank Miss Anne Harrison for the histological preparations and Mr. David Andrews for the photographs.

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TWO CASES OF BILATERAL BRANCHIOGENIC CYSTS OF THE NASOPHARYNX

By OMAR H. SHAHEEN (London)

Introduction

Previous articles have provided detailed information on the subject of branchiogenic cysts of the nasopharynx, with regard to site of origin, histological features and embryology.

Taylor and Burwell (1954) reviewed the past literature, adding four cases of their own, two of which were of bilateral cysts.

Wild and Fruhling (1953) reported on a unilateral cyst, which was not specifically labelled as branchiogenic, but had many of the features of that type of cyst.

Caliceti and Silimbani (1955) described the case of a unilateral cyst, resembling the branchiogenic type, but arising at a higher level, and classified by them as "dysembryogenetic". Mills (1959) recorded a typical case of a unilateral cyst.

Two cases of bilateral cysts are now presented, and are termed branchiogenic, although the site of origin of the cysts in each case differs from that previously described, in that it is situated at a higher level.

Case I

F.V.M., a male aged 18, complained on *25.2.58* of bilateral nasal obstruction, with greenish nasal and post-nasal discharge for four years. Tonsils and adenoids had been removed ten years previously.

Anterior rhinoscopy showed pus in both nares, and posterior rhinoscopy revealed a "swelling", the characteristics of which could not be determined owing to the presence of over-lying pus. The left antrum was opaque radiologically but X-ray of the base of the skull was normal.

On *4.3.58* an examination of the nasopharynx under anæsthesia revealed two symmetrical fleshy but firm swellings with pink granular surfaces, occupying the postnasal space, and separated by a median cleft up to the midline of the vault. The swellings arose from each side of the vault, the fossae of Rosenmüller and the lateral wall behind the Eustachian openings.

Several biopsy specimens were taken, whereon the swellings collapsed, and their cystic nature became obvious. Histologically, the wall of each cyst was made up of fibrous tissue. The internal lining epithelium was squamous, varying in thickness, being stratified in some parts and reduced to one or two layers of flat cells in others. No lymphoid tissue was present in the cyst wall.

There has been no recurrence of the cysts to date.

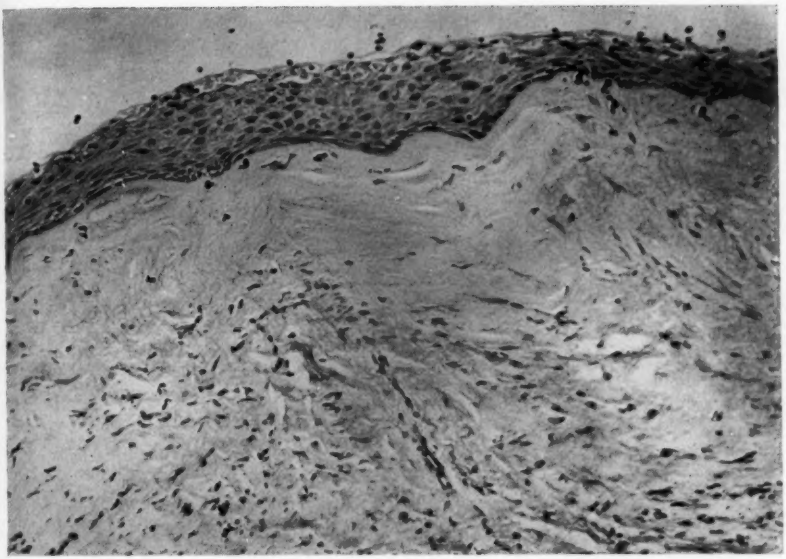

FIG. 1.

The cyst wall from Case I, showing stratified squamous epithelium, and the absence of lymphocytes (×152).

CASE II

A.C., a male aged 59, complained on *12.9.59* of progressive deafness in the left ear for one year, and clear nasal discharge for twenty years.

Inspection of the oro-pharynx revealed the lowermost part of a smooth, pale, glistening swelling, hanging down behind the soft palate on the right side.

Posterior rhinoscopy showed that the nasopharynx was occupied by two swellings, separated by a median cleft, and differing in size, shape and colour The one on the right, was pedunculated and considerably larger and paler than the left one. It occupied the whole of the right side of the nasopharynx and obscured both the right Eustachian opening and the right choana. This was the swelling that hung down below the free edge of the soft palate. That on the left, was a dome-shaped sessile, pink, smooth swelling, occupying the left hemi-vault, and obscuring the left Eustachian aperture.

The tympanic membrane and the hearing on the right side were normal, but the left membrane was retracted, and a severe conductive deafness was present on that side. A diagnosis of branchiogenic cysts was made.

At operation on *29.9.59*, the nasopharynx was approached by detachment of soft palate from hard. The cystic nature of the swellings was confirmed. They both arose partly from the vault, the fossa of Rosenmüller and the lateral wall behind the Eustachian orifice. The right-sided swelling had a well-marked circular constriction about its middle. Both cysts were successfully excised. The patient made a good recovery, and the hearing was completely restored

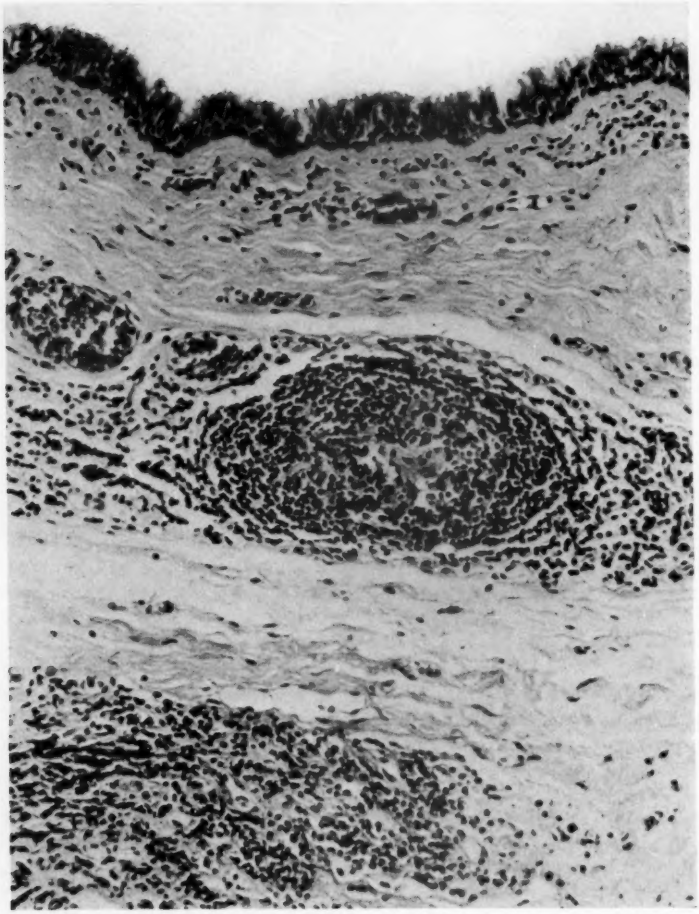

FIG. 2.

The cyst wall from Case II showing pseudo-stratified columnar ciliated epithelium, and a clump of lymphocytes (× 185).

in the left ear. Histological examination of the cysts showed a wall of fibrous tissue containing isolated clumps of lymphocytes, and an epithelium which varied in appearance, being transitional in places and reduced to a single layer of flat cells in other, but for the most part it was pseudo-stratified columnar ciliated epithelium.

Discussion

The usual site of origin of branchiogenic cysts of the nasopharynx lies between the upper pole of the tonsil and a point just below or behind the

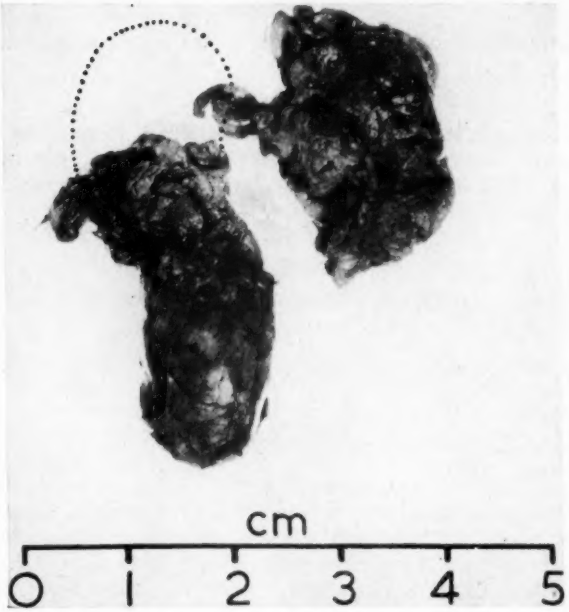

FIG. 2(*a*).

The two cysts from Case II. The upper half of the right-sided cyst is missing, but its position is indicated by the dotted line. There is a constriction around its middle.

Eustachian aperture. In the cases presented the cysts arose at a higher level. However, in view of the bilateral and symmetrical site of origin, the probability is that they are of the branchiogenic type. It is of interest to note in this connection, that in one of the cases of bilateral cysts recorded by Taylor and Burwell, a "tail", or solid cords was found in continuity with the upper pole of each cyst, inclining upwards, forwards and medially along each lateral wall and across the vault to meet in the midline of the roof just behind the septum. This suggests the possibility that branchiogenic cysts could arise anywhere along a line joining the upper pole of the tonsil with a point in the midline of the vault of the nasopharynx. This line would incline upwards from the upper pole of the tonsil, behind the Eustachian orifice, along the fossa of Rosenmüller and finally deviate medially and slightly forwards to the midline of the vault.

Two histological features that are unusual in the cases presented are the absence of lymphocytes in the cyst wall in the first case, and the variation in the appearance of the epithelium in the second one.

The constriction in the right-sided cyst of the second case was presumably due to pressure from closure of the nasopharyngeal isthmus around the middle of the swelling.

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Summary

Two cases of bilateral branchiogenic cysts of the nasopharynx are presented.

Acknowledgements

I wish to thank Mr. L. F. W. Salmon and Mr. P. Reading for allowing me to publish the two cases. I am also grateful to the Department of Medical Illustration, Guy's Hospital and Mr. A. Finch for the photographs and photomicrographs.

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TUMOUR OF GLOMUS JUGULARE

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Case Report

The first detailed anatomical description of the glomus jugulare was given by Guild, 1941. Further details were given by him in 1953. He found that one or more of these bodies were normally present along the course of the tympanic branch of the glossopharyngeal nerve, and the auricular branch of the vagus nerve, especially around the portions of these nerves in the jugular fossa. Another site for it is in association with the tympanic plexus at the tympanic promontory.

The histological structure of the glomus jugulare is similar to that of the carotid body. It is formed of rounded epithelioid cells arranged in alveolar clusters, supported by a stroma of collagen tissue rich in blood vessels. Like the carotid body, it is supplied by the glossopharyngeal nerve and the ascending pharyngeal artery. On basis of this similarity in structure, nerve and blood supply, a similar function has been also suggested, that the glomus jugulare is chemoreceptor.

The first tumour arising from the glomus jugulare was reported by Rosenwasser, 1945. Following Rosenwasser, several reports (Bickerstaff and Howell, 1953; Henson *et al.*, 1953; Lattes, 1950) have been published, until 1955 when Huppler *et al.*, were able to collect 100 cases from the literature.

As the early symptomatology of the tumours of the glomus jugulare is otological, otologists only were interested in that subject for a considerable time. Little interest was directed to the neurological complications of these tumours, until recently. Henson *et al.*, 1953, reviewed the literature, collected 77 cases, and added 6 cases of their own. They found an evidence of intracranial extension in 40 per cent. of such cases. Bickerstaff and Howell, 1953 analysed 90 cases reported in the literature, and found that 68 per cent of cases showed paralysis of one or more of the lower cranial nerves, in addition to disturbances of the auditory functions.

The first case reported in Egypt was by one of us, Hassan, in 1957. Our present case is reported as the second example of these comparatively rare tumours seen in our Neurology Department. It shows, more or less, the typical clinical picture of glomus jugulare tumours; unlike the first case, the interesting features of which are mentioned later in the discussion of the present case. Moreover, our second case followed in its progress the classical sequence of events followed by such tumours, and showed the necessity of a generous biopsy for the diagnosis of the microscopical pathology.

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Case Report

Patient, "F.A.A.", female aged 45 years, was admitted to Ain Shams University Hospital on *16.12.1958*, complaining of profuse paroxysmal bleeding from her right ear, and tinnitus in the same ear. She also complained of inability to close her right eye properly. Her complaint began 3 years before admission, by throbbing tinnitus in the right ear and throbbing headache every now and then, mainly in the right temporal and frontal regions. Two years after the onset, she noticed deviation of her mouth to the left side, with inability to close her right eye properly. Food began to accumulate in her right cheek. She received physiotherapy for this facial palsy, but the condition did not improve. One year later, she noticed paroxysmal profuse bleeding from her right ear with progressive right-sided deafness.

On examination: She was of moderate build and clear mentality. Visual acuity, field of vision and fundi were normal. Ocular movements and pupils were normal, there was no nystagmus. There were no signs of affection of trigeminal nerves. The right face showed evidence of lower motor neurone lesion, with loss of taste on the anterior two-thirds of the right half of the tongue. Hearing was diminished on the right side, with bone conduction better than air conduction. No bruit was heard over the mastoid, but a loud bruit was heard on the right carotid bifurcation, which was visibly pulsating. No enlarged lymph nodes were detected. The uvula deviated towards the left side, and there was right hemiatrophy of the tongue, with deviation to the right side on protrusion. Motor system, deep and superficial reflexes and sensations were all normal.

E.N.T. Report

(Given by Prof. Ali El-Mofti, Ain Shams University.)

Nasopharynx and nose: were clinically free.

Larynx: there was partial paralysis of the right vocal cord which was fixed in the middle line.

Ears: left ear was free. Right ear showed a mass reaching to the bottom of the concha and filling the whole external auditory meatus, not adherent to the walls of the meatus. By gradually increasing the pressure in the external auditory meatus, the mass showed pulsations. The mass was soft, necrotic, offensive, friable, bluish and bled profusely.

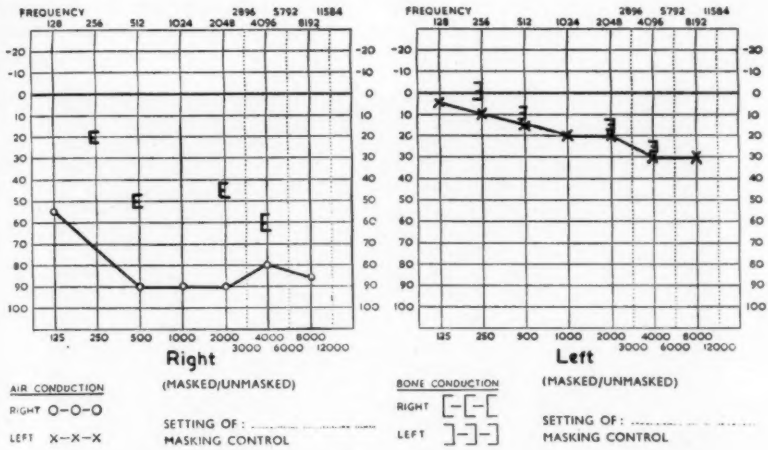
Audiometry: left ear was within normal limits. Right ear showed marked drop of both air and bone conduction. The deafness was found to be mainly conductive, as the bone conduction was better than air conduction in all the frequencies, but as hearing loss was more than 60 decibels, nerve deafness could not be excluded. (Audiogram.)

Vestibular tests: Calorimetric tests were not feasible due to the tumour occupying the whole of the external auditory meatus. Galvanic and rotation tests were not done.

Blood Picture: Hb: 100 per cent., *R.B.C.:* 3,890,000, *W.B.C.:* 12,000 *Diff. count: P:* 70 per cent., *L:* 28 per cent., *M:* 1 per cent., *E:* 1 per cent., *B:* 0 per cent.

Bleeding time: 3 min. *Coagulation time:* 10 min. *Blood platelets:* 240,000/c.mm. *W.R.* negative.

C.S.F.: clear, colourless, showing no clot or deposit. Proteins 30 mgm., chlorides 320 mgm., cells 4/c.mm., lymphocytes. *W.R.:* negative.



Audiogram

Radiographic studies: plain X-ray of the skull showed destruction of the right mastoid with haziness of right mastoid cells and sequestration (Fig. 1). The left mastoid was normal (Fig. 2). The right temporal bone showed destruction of its petrous portion (Fig. 3). There was no change in the foramina of the skull base.

Carotid angiography: showed normal filling of the right internal carotid system (Fig. 4). The external carotid system was not visualized.

Thyroid gland: was enlarged and nodular, but I^{131} uptake was found normal, and scanning of both mastoid regions showed no evidence of increased uptake there.

Blood pressure: was 210/90 and the report of the cardiology section was one of systolic hypertension, with no affection of the heart.

Biopsy: was taken after control of the high blood pressure, and antibiotic and hygroscopic aural drops. The report was given by Dr. I. Friedmann, Pathologist at the Institute of Otolaryngology, London, and was as follows:

"Section shows neoplastic tissue underneath the necrotic squamous epithelium of the external auditory meatus. The surface of the tumour is partly covered by inflammatory granulation tissue. The tumour consists of round or polygonal cells resembling epithelial cells. The cytoplasm of the cells is vacuolar, the nuclei are round and darkly stained. The cells are arranged in aggregates or groups separated by fibrous tissue. The characteristic alveolar pattern is best shown in the central parts of the section. The tumour does not appear to be very vascular in the sections examined, but there is a number of small rather compressed capillaries present (Fig. 5). Tumour of glomus jugulare."

Discussion

Our case represents almost all the clinical features of tumours of the glomus jugulare. The tumour is more common in females (Bickerstaff and Howell, 1953; Capps, 1944; Henson *et al.*, 1953; Siekert, 1956). The age at onset; although variable from two up to sixty five years, yet the maximum age incidence is

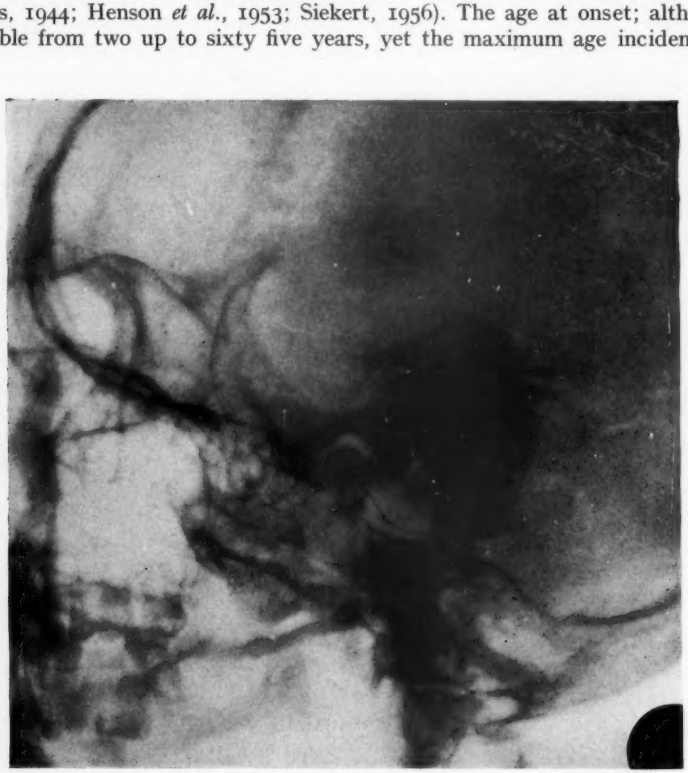


FIG. 1.

Plain radiography showing destruction of the right mastoid with haziness of the right mastoid cells and sequestration.

between forty and sixty (Siekert, 1956). The onset with aural symptoms; tinnitus, deafness, and more important, bleeding from the ear, is the mode of onset in most of the cases already reported. The sequence of events is rather classical in our case, i.e. after aural symptoms the facial nerve was involved and was followed by paralysis of the lower medullary group of cranial nerves without signs of increased intracranial tension or long tracts affection. However, in contradistinction to the usual classical description the lower medullary group of cranial nerves (Xth and XIIth) were involved in a relatively short period after the appearance of aural symptoms in our patient. But, although many authors have stressed the fact that neurological symptoms

and signs (other than VIIIth and VIIth cranial nerve involvement which usually appear early) appear a long time after aural symptoms, yet this is not an absolute rule. Some of the cases reported by Siekert, 1956, showed intracranial manifestations before the appearance of their aural symptoms, and other cases showed evidence of intracranial extension concurrently with aural symptoms. Henson *et al.*, 1953 (Case No. I) reported palatal and hypoglossal

FIG. 2.
Plain radiography showing left normal mastoid.

paralysis appearing fifteen months after the onset of otological symptoms. In a very recent case reported by Shreekumar and Rosenbluth, 1959, neurological signs other than VIIIth and VIIth nerve palsies, occurred in a period of less than three months after the onset of aural symptoms.

The variation in the clinical picture seems to depend on the origin of the tumour. Tumours which arise from the glomus structure around the jugular bulb, are more liable to extend intracranially earlier than those arising from the glomus structure in the tympanic cavity.

All the neurological signs present in our case, have been previously reported. Other signs which are lacking in our case are less common, e.g. IIIrd, IVth, Vth

and VIth nerve palsy. This occurred in the first case reported by one of us Hassan, 1957. This latter case showed anterior extension on the tumour producing primary optic atrophy and proptosis, and a pathological fracture of the neck of the femur suggesting distant metastasis. Recently (April, 1960), follow up examination of the same case showed congestion of the neck veins denoting

FIG. 3.

Plain radiography of the base of the skull showing destruction of the petrous portion of the right temporal bone.

invasion of the jugular vein by the tumour. Angio-invasiveness has been previously reported in nine cases with intracranial extension in six of them (Shreekumar and Rosenbluth, 1959). The vessels invaded were the jugular vein, the sigmoid sinus, the transverse sinus, the petrosal sinus and the cavernous sinus.

Horner's syndrome, cerebellar signs and contralateral pyramidal signs may be rarely found. Papillœdema is also rare (Hierons, 1954).

The plain radiographic changes in the form of destruction of the mastoid and petrous temporal bone, are found to occur in about half of the cases of glomus tumors with intracranial extension (Siekert, 1956).

The carotid arteriogram is normal in our case because the internal carotid system only was filled. The frequent failure of the tumour to show in the ordinary carotid arteriogram was commented on by Henson *et al.*, 1953. This was supposed to be due to the fact that the external carotid artery, and less commonly the vertebral artery, supply the tumour, but rarely the internal carotid artery supplies it. Our patient did not accept the performance of another arteriogram for the external carotid system. Carotid arteriography

FIG. 4.

Right carotid angiography showing normal filling of the right internal carotid system. The external carotid system was not visualized.

in our first case (Hassan, 1957) showed a diffuse shadow extending from the sellar region to the neighbourhood of the mastoid bone, and uppermost part of the neck.

Confirmation of the diagnosis by biopsy was done. The biopsy taken has to be generous, and not taken from the surface only as the latter may not show any evidence of the tumour but only that of infected granulation tissue. As a matter of fact, the pathological report in the first biopsy of this case was; "infected angiomatous granulation tissue". The second biopsy, which was more generous showed definite evidence of tumour cells. The presence of epithelioid cells, with vacuolar appearance of the cytoplasm, and the alveolar arrangement of the cells in parts of the tumour, all these pathological features, are rather similar to the previously reported cases of tumours of glomus jugulare.

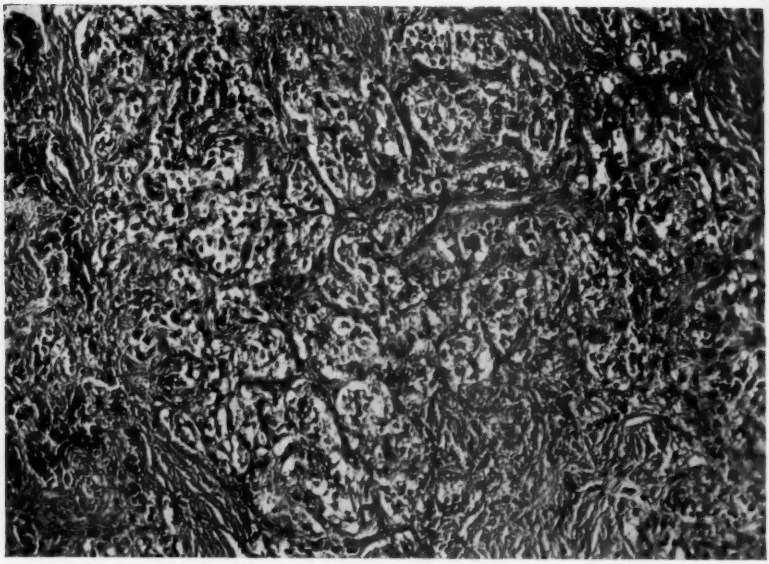


FIG. 5.

Representative area of the Tumour showing the characteristic Alveolar Pattern: the Epithel'oid cells are arranged in groups separated by Fibrous Tissue. H. and E. ×200.

Summary

1. Except for the short history, this case shows the classical clinical picture of tumours of the glomus jugulare.

2. It started with otological symptoms followed by profuse bleeding from the ear, peripheral facial palsy and involvement of the lower cranial nerves.

3. Plain X-ray showed destruction of the right mastoid bone.

4. Biopsy showed cells with a vacuolar cytoplasm. The cells are arranged in alveoli in certain parts and aggregates of the cells are separated by fibrous tissue, containing compressed blood vessels.

Acknowledgements

We thank Dr. I. Friedmann (Department of Pathology and Bacteriology, The Institute of Laryngology and Otology, London), for his report on the pathology of this case, and Prof. A. El-Mofti (Ain Shams University, Cairo) for his E.N.T. report.

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CLINICAL NOTE

IN DEFENCE OF TRANSILLUMINATION

By A. TUMARKIN (Liverpool)

In the January issue of this Journal, Mr. M. E. N. Smith asserts that "Transillumination is out of date. Its continued use without X-ray is a cause of loss of life and probably amounts to criminal negligence with no defence in Law".

The arguments used to support this sweeping conclusion would seem to be curiously lacking in clinical understanding and indeed in logic.

The first case quoted is that of a young woman whose right antrum proved dull on transillumination. Repeated wash outs revealed pus but the underlying carcinoma was not recognized until very much later.

This surely cannot justify a criticism of transillumination. That test had revealed all that an experienced clinician could expect from it—namely dullness leading to proof puncture which entirely justified the transillumination by revealing pus. The subsequent conduct of the case may or may not be open to criticism but it certainly does not justify this denigration of the initial transillumination.

The second case quoted by Mr. Smith is even less relevant, indeed it seems a complete "non sequitur". Once more "sinusitis" was diagnosed by transillumination, and once more there was no adequate response to repeated lavage. In this case an X-ray was taken "relatively early" but the "neoplastic erosion" was misdiagnosed as "osteomyelitis" and the "lavages continued unabated".

Once more we must ask how can this lamentable episode justify denigration of transillumination? That test had amply justified itself by leading to a recognition of antral pathology. The subsequent tragedy was in no way related to the transillumination.

Mr. Smith's thesis appears to be that transillumination should never be performed and that it should be replaced by routine X-ray. Yet his cases prove:

(1) That transillumination combined with adequate clinical judgement can be entirely adequate. In both cases the failure of proof puncture and the persistence of foul pus called for open exploration without delay.

(2) That in the absence of clinical acumen *no test* can be of any avail. Transillumination in the second case had entirely justified itself by indicating antral pathology, X-rays *were* taken "relatively early" but even so the correct diagnosis was not made.

Mr. Smith demands that the clinician shall give his patients all the advantages of modern scientific tests. No one can deny that it seems probable that the most imposing battery of tests will fail unless the clinician brings to bear on his cases a finer clinical acumen and a more scrupulous logic than are apparent in Mr. Smith's own arguments.

GENERAL NOTES

THE YEARSLEY MEMORIAL LECTURE

The Yearsley Memorial Lecture will be given at the Royal Society of Medicine on Friday, March 3rd, at 5 p.m. The lecture will be read by Mr. J. D. K. Dawes on behalf of Dr. Louis Verhoeven, who is unfortunately ill. The subject of the lecture is "The examination of a chronic ear".

VIIème CONGRÈS INTERNATIONAL O.R.L.

Le VIIème Congrès International O.R.L. se tiendra du 23 au 29 juillet 1961 à Paris, à la Nouvelle Faculté de Médecine, Rue des Saints-Pères, sous la présidence de M. le Professeur Aubry.

Trois très importants rapports seront discutés:

1°) Les acquisitions récentes en pathologie et en thérapeutique de l'otosclérose.
Rapporteurs: MM. les Professeurs Sourdille, Sercer and Krmpotic, Weber, Ardouin, Larroude, Ferreri and Schambaugh.

2°) Indications et résultats après 5 ans, de la chirurgie et des associations radio-chirurgicales dans le traitement des cancers du larynx et de l'hypopharynx.
Rapporteurs: MM. les Professeurs Pietrantoni and Agazzi, Alonso, Casadesus, Chevalier-Jackson, Leicher, Leroux-Robert, Ormerod.

3°) Rapports entre l'allergie et les infections du nez et des bronches.
Rapporteurs: MM. les Professeurs Van Dishoeck, Clerici, Hlavacek, Voohorst, Mayer, Laskiewicz, Teracol.

De nombreuses réunions et festivités sont envisagées pour les Congressistes et leurs épouses. A la suite du Congrès, des excursions auront lieu; elles seront proposées dans un avant-programme et réalisées par les soins des Wagon Lits Cook, 22 rue de l'Arcade, Paris, qui sont notre agent officiel habilité pour les transports et réservations d' hôtels.

Pour tous renseignements, écrire au Dr. Henry Guillon, Secrétaire Général, 6 avenue Mac-Mahon, Paris.

THE INSTITUTE OF LARYNGOLOGY AND OTOLOGY

330-332, Gray's Inn Road, London, W.C.1.

SHORT COURSES

for

CONSULTANTS AND SENIOR REGISTRARS

28th and 29th April, 1961.—Practical Aural Surgery Class.

General Notes

NON-MEDICAL COURSES

31st May, 1st and 2nd June, 1961—Hearing and mental assessment of the deaf child—for psychologists.

The number of entries is limited in each course. A detailed syllabus of the above can be obtained on application from the Secretary-Administrator.

SPECIAL LECTURES

Saturday, 6th May, 1961. Symposium beginning at 10 a.m.

Professor Otto Löwenstein, Birmingham. "The evolution of static and acoustic sense organs."

Dr. Hallowell Davis, St. Louis. "The relation of presbyacusis to noise-induced hearing loss."

Dr. Juergen Tonndorf, Iowa City. "Cochlear hydrodynamics."

UNIVERSITÀ DEGLI STUDI DI BOLOGNA
PREMIO INTERNAZIONALE "PIETRO CALICETI"

Announcement of the conditions for the International Prize Pietro Caliceti

The conditions for the "International Prize Pietro Caliceti" are herewith announced. The prize honours the memory of Prof. Pietro Caliceti, deceased distinguished Professor of Clinical otorinolaringojatrica of the University of Bologna.

The prize will be awarded to an Italian or foreign graduate in Medicine and Surgery, who during the four-year period of time (November 1, 1956 to October 31, 1960) has or will publish a scientific work of unquestionable value and of recognized originality in the field of otorinolaringologia.

The prize consists of: (*a*) a gold medal, minted from the original mould used by Mrs. Inez Misley, the widow Caliceti; (*b*) a cash prize of 1.000.000 Lira; (*c*) Diploma testifying to the receipt of the prize itself.

The Committee of Judges will be composed of: (1) The Dean of the Faculty of Medicine and Surgery of the University of Bologna. (2) The Professor of Clinical Otorinolaringologia, of the University of Bologna. (3) The President of the Italian Society of Otorinolaringologia. (4) The Professor of Clinical Surgery of the University of Bologna. (5) The professor of Human Physiology of the University of Bologna.

If in the final judgement of the Committee, none of the works presented by the competitors demonstrates outstanding value and originality, they may decide not to award the prize.

By April 10, 1961 the competitors must send to the "Segreteria dell'Università di Bologna" the following documents: (*a*) an application addressed to the Magnifico Retoret dell' Università degli Studi di Bologna; (*b*) a curriculum vita; (*c*) six copies of each scientific work submitted for the examination for the prize (five of these will go to the members of the Committee of Judges).

In addition, the Committee of judges may give consideration in awarding the prize to informations and data submitted by the above mentioned date by foreign or Italian Universities or by the three societies of Otorinolaringologia: Italian, Latin, International.

TEMPLE UNIVERSITY SCHOOL OF MEDICINE

PHILADELPHIA 40. PA.

A 'Short course in Audiology" is offered several times each year by the Section of Audiology, Department of Otorhinology, Temple University School of Medicine, Philadelphia 40, Pa.

The course will be one week in length and will encompass theoretical and clinical aspects of audition. Hearing testing and interpretation will be stressed. Office procedures, equipment and Industrial Audiometry will be discussed.

Further information may be obtained by writing to Philip E. Rosenberg, Ph.D., Director, Section of Audiology, Temple University Medical Center, 3401 North Broad St., Philadelphia, Pa.

ROYAL COLLEGE OF SURGEONS
OTOLARYNGOLOGY LECTURES

THE following lectures, arranged jointly by the Royal College of Surgeons of England and the Institute of Laryngology and Otology, will be delivered in the Lecture Hall of the College on Thursday afternoons at 5.30 p.m.

1961

March 2nd. PROFESSOR L. P. GARROD. "Principles of Antibiotic Therapy in Surgery."

May 4th. DR. R. BODLEY SCOTT. "Blood diseases in relation to Otolaryngology."

UNIVERSITÉ DE BORDEAUX
—FACULTÉ DE MÉDECINE ET DE PHARMACIE
CLINIQUE OTO-RHINO-LARYNGOLOGIQUE

COURS DE PERFECTIONNEMENT

Du Professeur G. Portmann

DU LUNDI 10 AVRIL AU SAMEDI 22 AVRIL 1961

Les inscriptions sont à adresser à: M. le Professeur G. Portmann, 45, cours Maréchal-Foch, Bordeaux

DROIT D'INSCRIPTION: 300 NF.

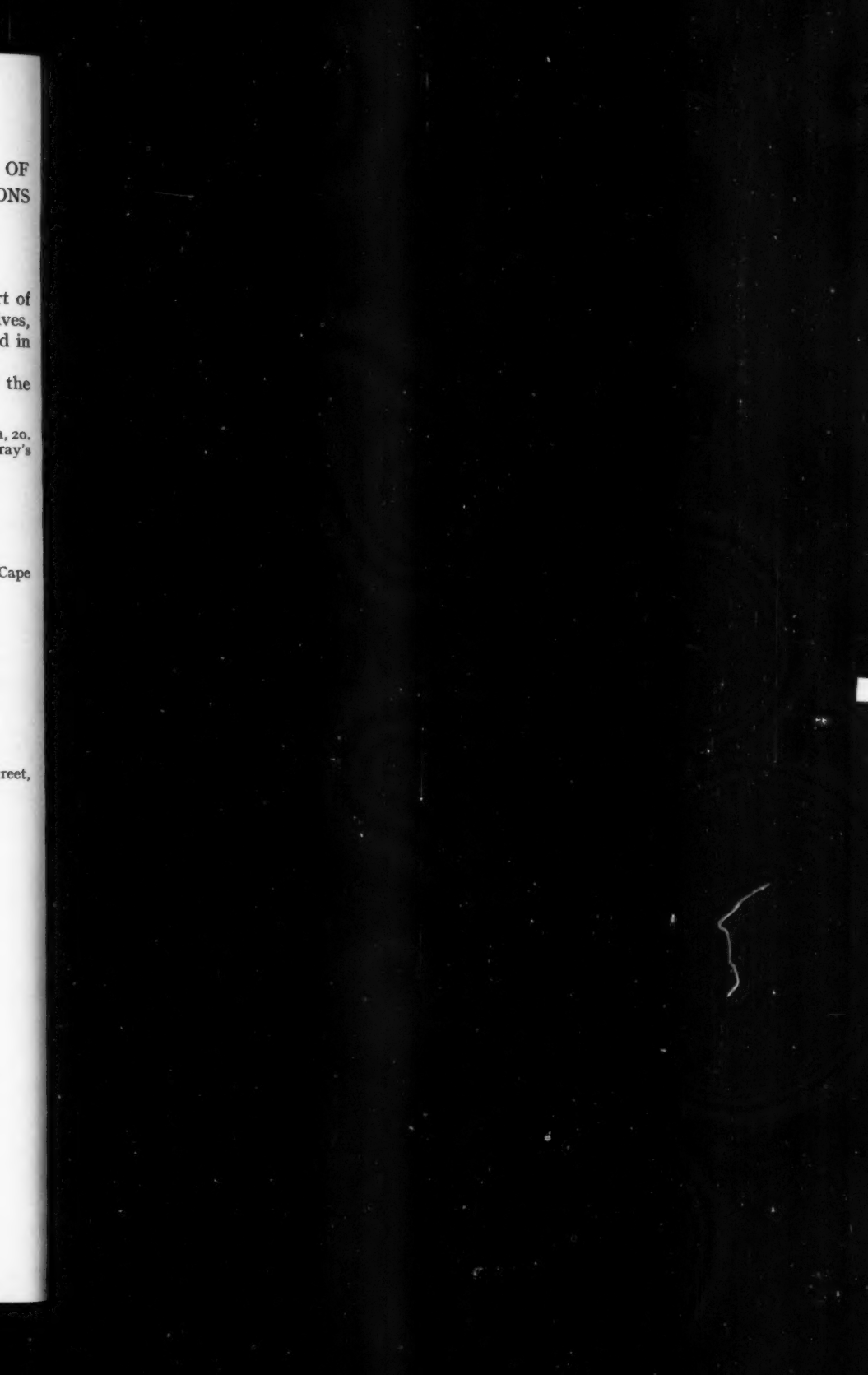
A verser a la Faculté de Médecine, Au Début des Cours.

General Notes

EXAMINING BOARD IN ENGLAND BY THE ROYAL COLLEGE OF PHYSICIANS OF LONDON AND THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

Diploma in Laryngology and Otology

The Secretary reports to the two Royal Colleges that at the First Part of the Examination held in December, 1960, 54 Candidates presented themselves, of whom 29 passed; and that at the Second Part of the Examination held in December, 1960, 40 Candidates presented themselves of whom 25 passed.

The following 24 Candidates, having complied with the regulations of the Board, are eligible to receive the Diploma, viz.:

Bokins, John	9 Hawthorn Park, Handsworth Wood, Birmingham, 20.
Atyle, Michael James	Royal National Throat, Nose and Ear Hospital, Gray's Inn Road, W.C.1.
Edwards, William Glyndwr	12A Kirkdale, S.E.26.
Ferris, Keith Parham	37 The Priory, S.E.3.
Gill, Balraj Singh	Ophthalmic Hospital, Maidstone, Kent.
Griffiths, Horatio Jonathan	60 Copthorne Park, Shrewsbury, Shropshire.
Gyaw, Christopher Pee	19 Build. 6, Lanthit Yeiktha, Rangoon, Burma.
Hasnain, Syed Wahaj-ul	35A Preston Road, Westcliff, Essex.
Hayes, Arthur Vivian	11 Sandhurst Road, Vincent, East London, Cape Province, S. Africa.
Hilmi, Suhail Rasheed	66 Greencroft Gardens, N.W.6.
Hla, Maung San	13 Panchan Street, Sanchaung, Rangoon, Burma.
Jackson, James McGillivray	11 Claremont Place, Newcastle-on-Tyne.
Jayarajah, Samuel Charles	St. Peter's Hospital, Chertsey, Surrey.
Jog, Mohan Ganesh	Royal Hospital, Wolverhampton.
Landeg, Mervyn Russell	83 Sandown Road, Port Talbot, Glam.
Mathen, Kayyalakakathu Mathen	St. Mary Abbotts Hospital, W.8.
Morris, Thomas Michael Owen	Brambletye, Poundhill, Worth, Sussex.
Owen, David Roy	The Vicarage Flat, Wilstead, Bedford.
Ramachandra, Vaitilingam	240/4 Torrington Avenue, Colombo, 7.
Thar Hlaing	32 Bolton Gardens, S.W.5.
Tucker, William Nichol	c/o Bank of New Zealand, 1 Queen Victoria Street, E.C.4.
Wijeyaratne, Donald Stanley	Seethala Agara Estate, Dunagaha, Ceylon.
Wilson, Ian Irvine	16 Glenhill Close, Lichfield Grove, N.3.
Yorston, Robert Allan	Templehall, Longforgan, by Dundee, Angus.